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TABLE OF CONTENTS.

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PAGE

ORIGINAL ARTICLES—

Deaths from Hydatid Disease ..... *Sir Louis Barnett* 205
Hydatid Disease of the Brain, with Report of a Case ... *W. A. Hailes* 212
Tendon Grafting in Paralytic Squint ..... *E. Temple Smith* 219
The Suspensory Ligament of the Pelvic Viscera *W. E. A. Hughes-Jones* 227
Thrombocytopenic Purpura: A Clinical Study *F. Tidswell and R. B. Wade* 233
The Surgery of the Lens ..... *E. A. Brearley* 244
The Intracapsular Extraction of Cataract with Sinclair's Forceps *A. L. Tostevin* 249
Hæmatemesis due to Gastric and Duodenal Ulcer .... *C. Gordon Shaw* 254
Some Observations on the Pathology of Appendicitis .. *Alfred J. Trinca* 258
Experimental Unilateral Uretero-Intestinal Anastomosis: Three Years' Survival after Unilateral Uretero-Colostomy Followed by Opposite Nephrectomy ..... *A. Bolliger and P. N. Walker-Taylor* 268
Some Uses and Abuses of Lumbar and Ventricular Puncture *I. Douglas Miller* 271
Osteochondritis ..... *John Hoets* 275

SURGICAL TECHNIQUE—

A Method of Extending a Fractured Humerus ..... *R. Douglas Wright* 283
Procidentia Uteri ..... *F. A. Maguire* 285

CASE REPORTS—

Internal Hernia through Foramen of Winslow ..... *J. K. Davidson* 311
Neurofibromatosis Associated with Proptosis and Defect of the Orbital Wall ..... *A. Eisdell Moore* 314

EDITORIAL—

PAGE

Hydatid Disease and Hospital Records 319

SURGERY IN OTHER COUNTRIES—

An Apparatus for Use with an Artificial Anus 321
The Treatment of Cardiospasm 321
A New Suture Material 322
Hydatid Cysts Ruptured into the Peritoneal Cavity and the Resulting Membranes of Encystment 322
White Mice and Experimental Echinococcosis 323
The Surgical Treatment of Chronic and Extensive Duodenal Ulcer ... 323
Hydatid Cysts of the Spleen 325

REVIEWS—

Hydatid Disease of Bone 327
A New Journal on Hydatid Disease 328
Nerve Suture 329
Work at the Mayo Clinic 329
Corrigendum 330

NOTICES—

Seventh Australian Cancer Conference 330
New Developments in Surgical Equipment 330
The British Post-Graduate Medical School 330

EDITORIAL NOTICES 330

THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

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DEATHS FROM HYDATID DISEASE.[1]

By SIR LOUIS BARNETT,

Emeritus Professor of Surgery, University of Otago, New Zealand.

THE first major operation I ever performed on my own responsibility was on May 4, 1891, at the Dunedin Hospital. The case was that of an attractive little boy, aged four, whose health troubles were diagnosed as being due to a hydatid cyst of the liver. I opened, evacuated, marsupialized and drained a large cyst of the liver by the abdominal route and I thought the patient would do well. But it was not to be. My little patient developed post-operative lung complications and died fifteen days after the operation. Autopsy revealed that in addition to the hepatic cyst there was a simple cyst the size of a duck's egg in the right lung and a rather larger suppurating cyst in the base of the left lung, which had burst into a bronchus.[2] This experience gave me a jolt which I well deserved, but it was the beginning of a keen and abiding interest in hydatid disease.

MORTALITY STATISTICS.

Davies Thomas, writing over fifty years ago, gave the mortality after operation for hydatid disease in Victoria as 32·8%.

Vegas and Cranwell estimated the rate in their Argentine experience as 13·6%.

Vegas in his book, "Hydatid Cysts of the Lung in Children", estimated the mortality in young people as approximately 20%.

K. D. Fairley, writing in *The Medical Journal of Australia*, February, 1924, says that for the thirteen years ending 1921 there were 258 patients with hydatid disease treated in the Melbourne Hospital, of whom forty-six died in hospital, equalling 17·7%.

Fulton, writing in *The New Zealand Medical Journal*, December, 1926, reported ninety patients admitted to the Dunedin Hospital for hydatid disease in the twelve years ending 1925. Of these, eighty-two

[1] Read at the annual meeting of the Royal Australasian College of Surgeons, Melbourne, March, 1935.

[2] Registry, Number 407.

were treated surgically and fourteen died—an operative mortality of 17·1%. Seven patients were not treated surgically and all of these died in hospital.

In a statistical survey of hydatid disease in New Zealand covering a period of 43 years ending 1934 (*The New Zealand Medical Journal*, August, 1934), I found that 2,840 patients with hydatid disease were treated in the New Zealand public hospitals, and of these 276 died in hospital. Many other patients no doubt went home to die, for the full total of deaths in the whole Dominion for the same period was 534, and it is fallacious to calculate the mortality rate from hospital figures alone.

Making reasonable allowance for cases dealt with in private practice, for unrecorded and unsuspected cases and for cases recorded more than once, I estimate that the mortality rate of hydatid disease is approximately 15%.

The Hydatid Registry, recently instituted by the Australasian College, and of which I have the honour to be Registrar, at present (January 1, 1935) contains records of 71 fatal cases in a total of 428, and from a study of these and of numerous other cases of which I have brief notes, certain conclusions emerge which form the basis of this address. I submit to you a tabulation of these fatal cases, indicating the location of the offending cysts.

Cases Recorded in the Hydatid Registry of the Royal Australasian College of Surgeons up to the beginning of the Year 1935, showing Location of Cysts and Mortality.

Site.	Cases.	Deaths.
Liver	249	45
Other abdominal, mostly secondary to liver cysts	50	7
Lung	74	12
Muscles and fascia	21	1
Bone	14	1
Kidney	11	2
Brain	5	2
Spleen	2	1
Thyreoid	2	
Total	428	71 (16·6%) [1]

[1] At the beginning of the year 1936 the case records on file number 630 and their analysis will appear in the annual report of the Registry.

SEPSIS.

An investigation of these cases shows clearly that sepsis with thirty-one fatalities is by far the commonest cause of hydatid death, and it may be accepted as a fact that the infection responsible for the sepsis comes in the great majority of cases from within and not from without. Fistulous communication with bile ducts or bronchi or both is common in old standing cases, and may be regarded as the usual portal of infection. Sepsis may of course be introduced from without at the time of operation or subsequent dressings, and in rare instances may also be serious enough to cause death, especially where the cyst cavity or subsequent sinus is extensive, folded, tortuous and difficult to keep clean. My colleague, Dr. Fulton, regards such cases as comparable

to a discharging psoas abscess and as calling for the most thorough Listerian antiseptic treatment.

In the great majority of cases fatal from sepsis the cysts had been growing for many years—ten, twenty, thirty, forty years or more—and extensive degenerative changes had taken place in the cyst membranes and in the surrounding adventitia, such as fibrosis, calcification, patches of necrosis, and in liver and lung cysts erosion of the walls of tubular channels.

We have to remember that hydatid cysts, except when situated in vital regions like the brain and spinal cord, are remarkably latent in their development as far as manifest symptoms are concerned. Modern authorities agree that infestation has most likely occurred in childhood and that, as Dew puts it, the majority of hydatid cysts as seen clinically are nearly as old as their hosts.

When a cyst of the liver is located in the anterior region and produces an obvious bulging in the upper part of the abdomen before many years are passed, operation is performed at a comparatively early stage and the results are good. When the cyst is deep seated it grows unnoticed to old and degenerate age and commonly trespasses on the thoracic cavity. It often pushes through the diaphragm, invades the pleural cavity and lung, and opens into a bronchus through which the contents may be partially expelled. Such deep seated cysts, usually already infected when operated on, necessitate a transthoracic approach for the purpose of evacuating the cyst contents and draining the cavity. It is not to be wondered at that fatal septic complications are common in such cases, particularly pleuro-pneumonic and subphrenic suppurations.

In deep seated cysts of the liver, erosion of bile ducts always occurs sooner or later, with consequent septic contamination, for the bile in such cases is always more or less infected. Partial evacuation of the cyst contents through one or more of the larger ducts may occur, and there may be blockage with hydatid *débris* resulting in obstructive jaundice. Particularly grave complications in such cases result from infection of the walls of the ducts and blood vessels, leading to fatal septicæmia and pyæmia.

Can this large tally of deaths from sepsis be reduced?

I doubt if much can be done in this direction apart from earlier recognition, earlier surgical treatment and improvements in operative and post-operative antisepsis.

It is, as you know, possible to treat some cases of suppurating hydatid cysts successfully without drainage, but this, in my opinion, is not advisable in old standing suppurative cysts with thick, rigid and perhaps calcified walls. In these I think the prognosis, no matter what is done, is deplorably bad. Old standing, obsolescent cysts with thick and calcified walls, usually diagnosed rather unexpectedly by X ray examination, are best left alone, provided that they are not causing trouble as a result of septic infection.

SEPSIS AND LOWERED INTRACYSTIC PRESSURE.

A biological characteristic of the typical, healthy, univesicular cyst is the high tension of the contained fluid. This feature does not seem to have received adequate attention in the books or papers on hydatid disease; but it strikes me as a matter of paramount importance, and it has distinct bearings on the occurrence of sepsis as well as other complications.

If a hollow needle is inserted into such a cyst, the fluid spurts out with considerable force, for the intracystic pressure resulting from the vital secretory activity of the germinal layer or endocyst is definitely higher than the normal blood pressure.[1] As is now well known, this spurting of fluid through a diagnostic or therapeutic and diagnostic

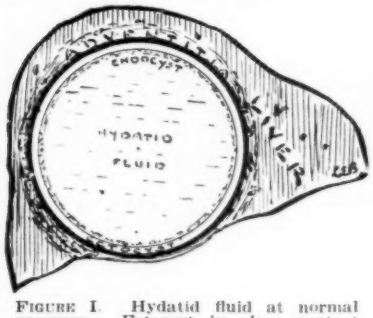

FIGURE I. Hydatid fluid at normal pressure. Ectocyst in close contact with the adventitia.

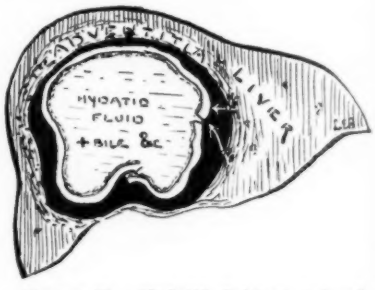

FIGURE II. Hydatid fluid at reduced pressure. Ectocyst falls away from the adventitia. Bile flows into the cyst cavity.

puncture may have serious and even disastrous consequences if it occurs within the thoracic or abdominal cavities (toxæmia, anaphylaxis, secondary echinococcosis *et cetera*).

The membranes of the hydatid cyst, the delicate granular endocyst as thin as paper, and the much thicker hyaline ectocyst have no organic connexion with the surrounding tissues of the host. These last named tissues are condensed into a fibro-cellular layer called the adventitia or pericyst, which transmits liquid nourishment to the parasite by a process of osmosis. The parasitic membranes are kept in close contact with the adventitia by the intracystic pressure of the hydatid fluid, just as the inside rubber bladder of a football is kept against its leather covering by inflation of air.

Surely this intimate contact of ectocyst with adventitia is of vital importance. If for any reason the intracystic pressure is lowered, the surrounding membranes will crumple more or less and will part company with the adventitia.

[1] Measurements of this intracystic pressure under varying conditions are now being conducted at the Dunedin Medical School. The only reference to its measurement that I have been able to find is the classic on hydatid disease by Davies Thomas, of Adelaide Hospital, in 1834. He estimated the pressure at 275 to 300 millimetres (11 to 12 inches) of water or 21 to 22 millimetres of mercury.

The ectocyst is a very friable layer and easily splits and becomes fragmented. Thereupon intracystic pressure is lowered by exosmosis through the adventitia. Fluids and also bacteria of varying virulence can then seep unhindered into the cyst cavity, and one has to remember also that bile ducts and bronchial tubes are embedded in the adventitia of liver and lung cysts respectively, and that their epithelial lined lumina are not always obliterated by the pressure of the growing cyst. Consequently when the ectocyst falls away from the adventitia, leakage of bile or bronchial secretion can take place into the cyst cavity, and, as is well known, these fluids in such circumstances are only too likely to be infected (see Figures I and II).

If the infection is of no moment, the result of the lowered intracystic tension is seen in a reproductive urge on the part of the endocyst, leading to the formation of daughter cysts, and sometimes in such profusion as to choke further vitality and end in obsolescence. If infection is of a higher degree of virulence, then suppuration and septic complications are brought about.

Blood vessels in the adventitia, being endothelial lined, are usually obliterated by the pressure of the growing cyst, but not always. Hence the lowering of intracystic pressure and the consequent separation of the ectocyst may lead to hæmorrhage, even fatal hæmorrhage.

HÆMORRHAGE.

In the list of 71 deaths there are 12 examples in which hæmorrhage was clearly responsible for the fatal event, and it was no doubt a contributory factor in other cases.

Fatal bleeding in hydatid cases may arise from three causes:

1. Mischance or error in operative technique whereby a large vessel is wounded. Fortunately this is a rare happening, but it does occur, and I dare say more often than records indicate. One case coming under this heading is recorded in the Registry (Index Number 182)—an operation traumatism causing uncontrollable bleeding when an unsuccessful attempt was made by puncture, cutting and burrowing to evacuate a deep seated lung cyst.

2. Oozing from blood vessels in the adventitia after the cyst has been evacuated of its contents. The Registry contains several instances of this occurrence, mostly associated with the presence of jaundice.

3. Erosion of a large blood vessel by pressure necrosis. There are three examples of this hopeless and helpless complication, the vessel concerned being the left common iliac artery (Number 306), the right common iliac artery (Number 265) and the portal vein (Number 25).

Dr. John O'Connor, of Buenos Aires, recorded a case operated on in 1895 in which he had cut and burrowed through 2·5 centimetres (an inch) of liver substance in order to reach a cyst. Terrific hæmorrhage resulted and ended fatally. He advised that in such deeply seated cysts the operation should be done in two stages: in the first firm parietal adhesions should be obtained, and later on opening into the cyst with a cautery knife should be made.

Sir George Syme, at the Australasian Medical Congress (British Medical Association) held at Dunedin in 1927, described two of his cases in which fatal hæmorrhage occurred. The first was the result of cutting through 1·8 centimetres (three-quarters of an inch) of liver substance in which the veins were enormously enlarged, and the second in which on opening a liver cyst the most terrific bleeding that he had ever seen took place and *post mortem* examination showed that this was due to erosion of the inferior *vena cava* as it lay in its groove in the posterior surface of the liver.

Herrera Vegas in his book, "Hydatid Cysts of the Lung in Children", refers to the case of Maximo Castro in which a cyst of the anterior mediastinum pushed the superior *vena cava* forwards, and at the operation this vessel was divided with immediately fatal results.

MULTIPLE ABDOMINAL CYSTS.

Multiple abdominal cysts are of common occurrence, and as is now well known, thanks to the work of Dévé, represent a secondary echinococcosis following dissemination of scolices from a burst or leaking primary cyst, almost invariably a liver cyst.

In the Registry there are at the moment 23 examples of this complication, with six deaths. Two patients (294 and 408) died from gradual exhaustion, two (175 and 406) from intestinal obstruction, one (232) from copious hæmatemesis, and one (278) from suppuration and hæmorrhage through a fæcal fistula. One of these patients (408) coughed up cysts and passed cysts by the bowel for years before he died.

CYSTS OF BOTH LUNGS.

Cysts of both lungs should be regarded with apprehension, for the operative prognosis is appalling. The Registry contains records of eight such cases, and six of the patients died after operation—a very heavy mortality indeed. Two of the fatalities occurred in my own practice. If collapse of one lung occurs as the result of operation, it is unlikely that the other lung, hampered as it is by the burden of one or more cysts, will be able to carry on the function of respiration adequately.

ANAPHYLAXIS.

Leakage or spilling of hydatid fluid within the body of the patient not only submits him to the well-known risk of secondary cyst formation, but also to the less well-known dangers of toxic absorption and anaphylactic reaction.

The milder manifestations of anaphylactic reaction are common enough, notably urticaria and slight pyrexia. Serious developments fortunately are rare, but may be not so rare as most of us have been in the habit of thinking. Notable contributions to our knowledge of this rather obscure subject have been published of late years by Dévé of Rouen, Dew of Sydney, and others.

Amongst the 71 fatal cases so far recorded in the Registry, there are five in which death might conceivably be attributed, wholly or

partially, to anaphylaxis of the major type. In three of these cases (264, 419 and 409) death occurred a few hours after rupture of a large liver cyst into the peritoneal cavity. In the fourth case (376) the surgeon did what he thought was a satisfactory operation for a suppurating liver cyst with subphrenic abscess, but a mysterious and unexpected collapse eight hours afterwards led to speedy death. The fifth case (159) was a patient of my own who had recovered by vomica from a suppurating pulmonary cyst, and on whom I operated for a liver cyst. He died on the operating table, I thought from the anæsthetic, but it might have been from anaphylactic shock.

FORMALIN AS A POSSIBLE DANGER.

Ill effects of formalin in the treatment of hydatid cysts have been alleged and contested by various writers. A colleague of mine at Dunedin, J. A. Jenkins, a skilful and experienced surgeon, who has long had some misgivings about formalin, sent me a record of a case of hydatid cyst of the liver in which the use of a 2·5% formalin solution on the protective packing and within the cyst cavity had been followed by sloughing in the wound area and cyst wall, septic infection, pyæmia and death. This is a very exceptional experience, however, and personally I have seen no harm resulting from my habitual use of a 2% formalin solution in accordance with the technique of Dévé and the limitations he counsels; nor have I had in my own practice any examples that I know of in which recurrence in the wound area has taken place.

After reading the unpleasant and sometimes alarming experiences in the use of formalin noted in some of the records, I felt convinced that Dévé's teachings on this subject should be more widely known, and in accordance with a request from the editor I have recently published a paper in *The New Zealand Medical Journal* of February, 1935, on formalin in the treatment of hydatid cysts.

OTHER CAUSES OF DEATH.

Other deaths recorded in the Registry are ascribed to: Cerebral cyst (Number 410), cerebellar cyst (387), hepatic insufficiency (396), cardiac embolism (416) resulting from the rupture of a liver cyst into the superior *vena cava*, thrombosis and embolism (26 and 213), bursting open of abdominal wound with extrusion of viscera (296 and 392), suppression of urine (21).

SUMMARY.

In this short paper I have drawn attention to the deaths, numbering 71, that have occurred in the 428 cases of hydatid disease recorded up to the present time in the Hydatid Registry of the Royal Australasian College of Surgeons. I have shown that sepsis from within is responsible for most of them, and I have explained the bearing of lowered intracystic pressure in this connexion. Hæmorrhage, multiple cyst invasion, especially of both lungs, and some uncommon causes of death are also mentioned.

HYDATID DISEASE OF THE BRAIN, WITH REPORT OF A CASE.

By W. A. Hailes,
Melbourne.

The case reported herein is of value not so much from its rarity as from the fact that it emphasizes many interesting features in the pathology, diagnosis and treatment of cerebral hydatid cysts. Even in countries such as this, cerebral cysts are seldom encountered, and the pathology as enunciated by Dévé and sponsored by Professor Dew is not well known. If this pathology is accepted, we must recognize two clinical types following primary or secondary infestation of the brain, the former resulting in solitary cyst formation, the latter causing multiple cerebral cysts.

The treatment of primary cysts, though beset with difficulties which were thought to be almost insuperable because of the age incidence and the risk of implantation cysts, affords much more hope of cure than does the treatment of secondary cysts. Advances in modern neurosurgical technique have overcome many of the difficulties previously emphasized in the treatment of primary cysts, but the risk of implantation cysts occurring or the effect of formalin on cerebral tissue cannot be disregarded. In some cysts the contents are aspirated and even ventriculography is performed before formalin can be injected.

The interesting features which this case emphasizes are: (i) methods of infestation—primary or secondary cyst formation and their significance, (ii) absence of adventitia, (iii) implantation cyst formation from scolices liberated at operation, (iv) unreliable results of serological reactions in cerebral cysts, (v) the possible rate of growth of implantation cysts in the brain.

Methods of Infestation.

Primary infestation in any organ occurs by the passage of the hexacanth embryo through the intestinal wall to the portal circulation, there to produce in a large percentage of cases an hepatic cyst.

Should the embryo traverse the portal circulation and not be arrested in the liver, it passes to the pulmonary circulation, there to produce a pulmonary cyst, or, traversing the pulmonary circulation, it is deposited in some portion of the capillary field of the systemic circulation. Should the embryo reach the internal carotid artery a primary cerebral cyst may result.

Secondary cerebral cysts occur as the result of rupture of fertile cysts into the left side of the heart. Scolices are then carried by the

systemic circulation to various parts of the body (Dévé's theory). If this pathology, which was first enunciated by Dévé and energetically sponsored by Professor Dew in this country, is accepted, it is at once evident that cases should fall into two clinical entities: (i) solitary primary cysts of the brain, (ii) multiple secondary cysts of the brain.

This pathology has not been universally recognized, even in countries such as Australia where hydatids are common, and, at the risk of correction, I would add is not universally accepted.

FIGURE I. Univesicular hydatid cyst of occipital lobe of the brain of a child, aged twelve years. From the Pathological Museum, University of Sydney. (Reproduced by permission from Dew's "Hydatid Disease".)

FIGURE II. Simple primary hydatid cyst of the frontal region of the brain of a child, aged eight years. From the Pathological Museum, University of Melbourne. (Reproduced by permission from Dew's "Hydatid Disease".)

In the case of primary cysts of the brain, all that is required is accurate localization and adequate removal of the cyst, which is a solitary cyst. Removal is easy because clinically there is macroscopically a complete absence of adventitia either from meninges, cerebral tissue or bone. This permits the collapsed cyst to be very easily removed with a dissector attached to the suction apparatus; there should be practically no scar after operation.

Some of the earlier surgeons in Australia emphasized the risk, in cerebral cases particularly, of implantation cysts in the operation area. This apparently happened in the case herein reported; and the rate of growth of the secondary implantation cyst is known, as it, like the primary cyst, was removed with the cyst wall intact, though collapsed, and it was after operation packed with cotton wool and preserved.

Primary cysts are nearly always cysts without daughter cysts, but containing scolices.

The cyst grows in the cerebral tissue till by pressure symptoms are produced. The cyst may impinge on the surface or reach the ventricle, and spontaneous rupture has been recorded.

FIGURE III. Multiple metastatic hydatid cysts, secondary to a cardiac hydatid. These cysts were all of the same size, that of a pigeon's egg, and were all simple univesicular cysts. The liver also contained a small cyst of about the same size in the upper part of the right lobe. The lungs were clear, but a small hydatid two inches in diameter, containing numerous small daughter cysts, was found in the wall of the left ventricle of the heart. (Reproduced by permission from Dew's "Hydatid Disease".)

According to Professor Dew, primary cysts are seven times as frequent in children as in adults, the incidence being 4·9% and 0·7%. The explanation given is that infestation with hydatids is most likely to occur in the early care-free years of childhood, when the risk of drinking infected water is less likely to be guarded against than in later years; and dogs are handled by children with little care or thought of result. Primary cysts therefore are most common in childhood.

Secondary cysts of the brain are entirely dependent for their age incidence on the period at which the primary cyst ruptures.

FIGURE IV. Degenerated hydatid cyst of the apex of the heart. Found at autopsy. From the Pathological Museum, Royal Melbourne Hospital. (Reproduced by permission from Dew's "Hydatid Disease".)

A primary cyst of the brain is associated with an hepatic cyst in 80% of cases.

SECONDARY CYST INFESTATION OF THE BRAIN.

I must say that I find it difficult to accept unreservedly the pathology that pictures secondary cyst infestation of the brain being due in almost all cases to the rupture of a cyst into the left side of the heart; that this accident could be one cause of secondary cerebral cyst formation is obvious; that it is the chief cause is possibly another matter.

Trauma to a cyst, whether by blows or muscular movement, are accepted as factors in daughter cyst formation and in the leakage of sufficient hydatid fluid into the surrounding tissues to render the patient anaphylactically sensitive. To accept this pathology means that when a cyst which is constantly being compressed by the cardiac musculature ruptures into the circulation, it sheds nothing larger than scolices, which are too small (160μ by 120μ) to cause an immediate cerebral lesion.

That is, neither minute daughter cysts nor shreds of hydatid membrane enter the systemic circulation to cause embolic phenomena; and acute anaphylaxis, though it has been recorded, is seldom mentioned. That this sometimes happens is proved; that it happens in the majority of cerebral hydatid cysts seems to me open to doubt. Unfortunately these cases are so rare that adequate *post mortem* statistics in this country are not available.

This pathological picture of secondary cyst formation has been enunciated to explain the majority of cerebral cysts in adults. Multiple cerebral cyst formations would seem to imply an accident, such as is mentioned with the liberation of many scolices into the systemic circulation. Several of the scolices must then enter the internal carotid artery.

The only other explanation could be that several hexacanth embryos made their way from the portal to the systemic circulation before arrest and development, and that more than one by chance reached the internal carotid artery.

Does the field where the hexacanth embryos or scolices are deposited influence growth or death?

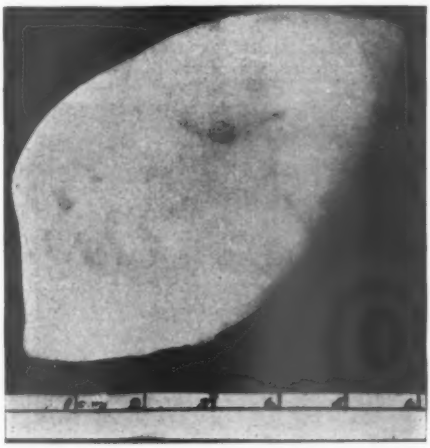

FIGURE V. Cyst removed at first operation.

RATE OF GROWTH.

The rate of growth of cerebral cysts is stated to be regular and comparatively rapid. Just how rapid that growth can be in the cerebral tissue is shown in this case, for, if we accept the recurrence as an implantation cyst, then the cyst attained a diameter of approximately 7·5 centimetres (three inches) in fourteen months.

Cerebral cysts are as a rule fertile with scolices within the brood capsule. Secondary changes do occur, but degeneration is not common. The cysts can reach a huge size, distorting the skull, rupturing into the ventricular system *et cetera*. By their pressure in silent or highly specialized areas of the brain they produce clinical syndromes similar in every way to those produced by non-infiltrating tumours.

I can find in the Australian literature no reference to the percentage incidence or rate of growth of implantation cysts in the brain.

DIAGNOSIS.

The diagnostic problem is the detection and localization of a cerebral tumour, and it would seem that serological reactions would prove its

nature. Serological reactions of course depend on the escape of sufficient antigen from a cyst to render the host sensitive, and it is well known that in the presence of primary cysts in the brain there is no response to the Casoni or complement fixation test. It would seem certain that when cerebral cysts are secondary to a ruptured cardiac cyst, there must be a response to the Casoni and complement fixation tests.

Unfortunately, in the presence of primary cysts the tests are valueless. It is greatly regretted that the serological reactions were not carried out in the following case till the day following operation; the patient's condition was otherwise fully investigated.

CASE HISTORY.

R.M., a female, aged forty-four years, was admitted to the Royal Melbourne Hospital under Dr. H. F. Maudsley with a history of severe frontal headache of six months' duration. The headaches, which were at first paroxysmal, became progressively worse and more persistent, particularly during the three months prior to admission. For the last two months her memory had begun to fail, while her responses to questions became uncertain and at times she exhibited a certain degree of aphasia. Three weeks before her admission to hospital her mother noticed that her speech was slurred; her writing began to deteriorate, while a progressive weakness in the right arm and leg developed, so that she could neither feed herself nor walk without assistance.

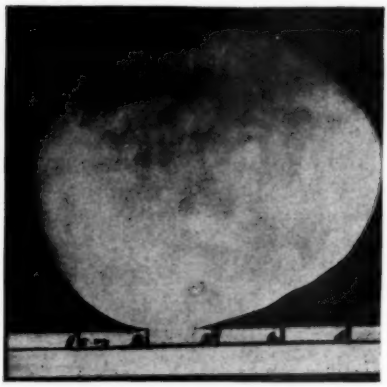

FIGURE VI. Cyst removed at second operation.

Neither gross vomiting nor gross interference with vision occurred, although she thought that her vision was not so good as formerly. Neither was there any history of hallucinations of smell or sight.

On examination, there were found no signs of disease in the thoracic or abdominal regions. Neither inspection nor palpation of the head revealed any abnormality. There was no ocular palsy, but there was a right-sided facial paralysis of the upper motor neurone type. The hearing appeared to be hyperacute, and there was some difficulty in swallowing, although the palate moved normally on both sides. In the optic disks venous congestion was present and a doubtful degree of papillœdema was noted. Owing to lack of cooperation and concentration, it was found impossible to determine the fields of vision. Radiological examination of the skull revealed nothing abnormal.

The superficial abdominal reflexes were present on both sides, but the left were much more active than the right. All the deep reflexes of the right side were much more active than those of the left, but there was no clonus, and plantar stimulation gave a flexor response on both sides. There were no signs suggestive of cerebellar involvement. The cerebro-spinal fluid was under excessive pressure, but was clear, and there was nothing abnormal detected therein. Neither blood nor cerebro-spinal fluid responded to the Wassermann test.

A diagnosis of tumour of the left cerebral cortex, involving the lower part of the motor cortex, was made, and operation was advised.

Operation was carried out, an osteoplastic flap being raised on the left side. This revealed in the left temporal region a subcortical swelling, which was discovered to be a simple hydatid cyst, about 7·5 centimetres (three inches) in diameter. The cyst was removed intact after evacuation, and the wound was closed without drainage. The fluid from the cyst contained a great number of scolices.

Convalescence was uneventful, and three weeks after operation the patient reported that the headaches had gone completely, and the facial paresis and speech difficulty were clearing up.

One day after operation a Casoni interdermal test was carried out, the response being a small wheal with a large area of erythema, but with no delayed reaction. The result would probably have been recorded as an equivocal reaction had not the history been known. No deviation of complement occurred with either cerebro-spinal fluid or blood.

The patient was readmitted to hospital on October 22, 1934. Her health had been reasonably good till July, 1934, when weakness of the right hand was noticed.

Six weeks later this was followed by weakness of the right leg, and this had been progressive, so that there was no voluntary movement in the right hand; there was some movement in the right leg, though the leg was very spastic.

Three weeks previously it had been noticed that the right side of the face was paralysed. The patient had complained of headaches, especially in the left frontal region.

Examination of the first to the sixth cranial nerves revealed nothing abnormal. Right lower facial paralysis was present, and deviation of the tongue and palate were noted. All the right-sided deep reflexes were greater than those on the left side. Patellar and ankle clonus was present on the right side. The plantar response on both sides was doubtful. The ocular fundi were normal. The fields of vision were normal.

The liver was not enlarged downwards; some questionable dulness was noted at the base of the right lung.

On November 20, 1934, the old osteoplastic flap was reopened, and a cyst was evident encroaching on the surface a little cranial to the site of the previous cyst. There was again a total absence of adventitia. The fluid appeared to be limited by the hydatid membrane alone, and unfortunately while arrangements were being made to photograph the cyst *in situ* the patient coughed and expelled the contents. The cyst was removed in one piece; the cavity was sponged gently with 10% formalin solution.

The cyst seemed to have made a bed for itself between the convolutions. The flap was replaced and the skull was closed. Progress was rapid and uneventful; the facial paralysis improved, and on December 5, 1934, five weeks after operation, the right arm and leg had so improved that the patient could walk reasonably well and use the left arm and hand for ordinary movements. All jerks were equal and active, and the clonus had disappeared.

On December 1, 1934, the patient was discharged to attend the outdoor department.

If the pathology outlined by Dévé is accepted, to which group does this case belong? Before the second operation Professor Dew considered it to be probably a primary cerebral cyst in an adult—a very rare condition, of which only two other probable cases are on record in Australia.

In excluding the possibility of the case being one of multiple secondary cysts, the following points must be emphasized:

1. That the second cyst was missed at the first operation is unlikely, since the second cyst was evident on the surface when exactly the same flap was turned down at the second operation.

2. Complete temporary recovery occurred and lasted for eight months following the first operation.

3. Definite serological reactions were absent, though it is admitted that tests were not made till after the first operation; but, surely, with rupture of a primary cyst all serological reaction would have been strongly positive.

4. That the serological reactions are changing now is not to be wondered at in view of the fact that on two occasions there has been an escape of some hydatid fluid at operation.

ACKNOWLEDGEMENT.

My thanks are due to Professor Dew for the interest he has taken and the help he has given me, and for his permission to reproduce illustrations from his book.

TENDON GRAFTING IN PARALYTIC SQUINT.

By E. Temple Smith,
Sydney.

It was in 1928 that I saw a paper in the *American Journal of Ophthalmology* by Rodin and Swett, in which the writers claimed to have obtained good abduction in sixth nerve paralysis by attaching to the insertion of the paralysed lateral rectus slips from the superior and inferior rectus tendons. Hummelsheim introduced the operation as long ago as 1907. Other successful cases have been reported by W. M. Sweet, R. O'Connor and S. R. Gifford in the same journal. It seems strange that so revolutionary a procedure should have attracted so little notice in English circles. I must admit, however, that I was a little incredulous

Figure I. Eighteen months after operation. Primary position.

Figure II. Eighteen months after operation, showing full range of movement to left.

myself. I did not see how it could act, either on physiological or mechanical grounds. Still, when a suitable case appeared, I looked up the original references and decided to try it.

The first case was that of a boy of fifteen months with a large left convergent squint. There was practically no movement towards the mid-line, owing to secondary contracture.

The history was that the child had had from birth an acute conjunctivitis, which lasted many weeks, and would have prevented early accurate observation. At about four months, the left eye was noticed by the parents to be turning in. The mother volunteered the statement that the child always, when looking to the left, turned the whole head round, while he did not do so on looking to the right, and this was easily demonstrable. Two of my colleagues saw the child, and we were unanimously agreed that the squint was a paralytic one.

I first did a subconjunctival tenotomy of the medial rectus to relax the contracture. This lessened the squint and the eye could now reach the middle line, but did not go beyond when the patient was looking to the left. Three months later the contracture had reappeared, and the child was pretty much as before the operation; so I decided to use a graft. This I did, and, a week later, performed tenotomy of the medial for a second time. The result, after the tenotomy, was dramatic. The child had now no squint in the primary position, and could move the left eye almost to the canthus. Eighteen months later the condition is the same. Figure I shows the eyes in the primary position, and Figure II shows the amount of abduction possible. I did not think of getting a picture of the condition before operation.

I hesitated about reporting this case, because I felt that, although it had all the marks of a paralytic squint, and did not behave like a

FIGURE III. Before operation, looking straight ahead.

FIGURE IV. After operation, looking straight ahead.

concomitant squint, it might be argued that it was only a case of "disuse". I believe that it was a true case of paralysis, but in so young a child it is obviously difficult to prove this conclusively. By way of control, some years ago I operated on a child with a paralytic squint, to see what would happen; I used the routine method of tendon lengthening and reefing. In the result, as might have been expected, there was no movement of the eye beyond the middle line. I have since declined to operate on such cases, except for a tenotomy to relax the contracture of the medial rectus. The future has more to offer.

The second case was that of a young woman of about twenty-five years, a doctor's wife. She had had a very large convergent squint from early infancy. She had been refused operation, she informed me, on account of the paralytic nature of the condition. The eye lay in the inner canthus, and could not be moved as far as the middle line, owing to secondary contracture of the medial. There was unequivocal paralysis of the left lateral rectus. The right refraction was + 2 diopters, with vision of $^6/_5$; that of the left was - 1 diopter, with vision of $^6/_{18}$ easily.

Here I did the same operation, but, after doing a complete medial tenotomy, I reattached the medial tendon slightly behind its original insertion (recession) at the same sitting. The immediate result was good, and it has continued to

FIGURE V. Before operation, looking to the left.

FIGURE VI. After operation, looking to the left.

FIGURE VII. Before, operation, looking well to left.

FIGURE VIII. After operation, showing full range of movement to the left.

improve. There is no convergence in the primary position, and parallelism is maintained with ease; and abduction, though not complete, is more than 45°. There is no diplopia. (See Figures III to VIII.)

A third case, which I owe to the courtesy of my colleague, Norman Gregg, was that of a boy of ten years, with a very pronounced congenital paralysis of the right lateral rectus. Curiously enough, there was no secondary contracture. But owing to the complete loss of tone of the lateral rectus there was a slight squint in the primary position, while, to avoid diplopia, the boy carried his head sideways, and progressed crab-fashion, which made him conspicuous among his fellows.

I did the same operation, with a slight recession of the medial. In this instance, with a view of lessening tension, I split the upper and lower tendons further up towards their origins than in the previous operations. This I believe to have been a mistake, but of this more later. At the end of a week there was not a vestige of right abduction, and I was disappointed. Week by week, however, increasing abductive power has developed, until, at present, a month after operation, there are 15° to 20° of abduction.

This case is not so dramatic as the others because the patient had not a pronounced squint in the primary position, as had the others. All the same, it is quite effective, as the boy, freed from diplopia, no longer carries his head sideways and is quite comfortable. And, though he does not get so much abduction as the others, he gets some. The reason that he gets less I believe I can explain later.

As the details of the operation in the original communication are somewhat meagre, a few remarks on the technique may be acceptable. The operation in the adult was done under cocaine and "Novocain" injection, but it was exceedingly difficult, as the patient had a small aperture and a deep-seated eye. Since the operation entails so much dragging on tendons, it must of necessity be painful. I shall therefore use general anæsthesia in future. After inserting an anchor stitch at the limbus for abduction of the eye, make a circular incision four millimetres from the corneal margin, reflect the conjunctiva with its underlying tissues from the tendons, and define each. Using a fine Stevens's squint hook, passed from behind forward, split off a third of the superior and inferior tendons by pushing the hook well up towards the origin of each. Pass a fine silk stitch round each fasciculus while *in situ*, and tie the stitch close to its insertion. With a Graefe knife detach its insertion, and at the same time shave off, if possible, a tag of superficial scleral tissue. This prevents the ligature from slipping and also provides a better surface for adhesion at the new site. Pass the needle from each ligatured fasciculus beneath, respectively, the upper and lower edges of the lateral rectus, but do not tie them. Now completely divide the insertion of the medial rectus, putting a stitch through the central fibres. While tension is thus relaxed tie the stitches, already in position, behind the lateral insertion. The medial tendon can now be left free or reapplied in such a position as seems desirable. Four separate sutures will complete the operation. Tie up both eyes for a week, removing the conjunctival stitches in three days. (See Diagrams I and II.)

A point which further experience will decide is this. In cases with strong secondary contracture (but not when there is none, as in the last case mentioned), it might be advisable, after having divided the medial, to leave this tendon unattached, but with a stitch through its

belly, for reattachment if and where it seems desirable at the end of a week. This would allow complete relaxation during the healing of the grafted slips.

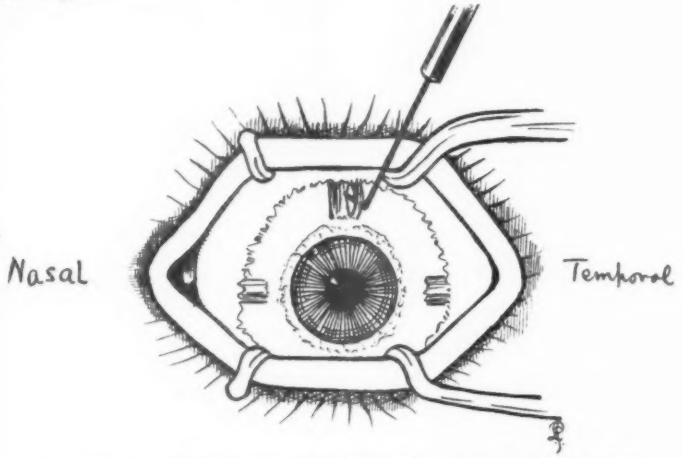

DIAGRAM I. Showing Stevens's fine hook splitting off fasciculus.

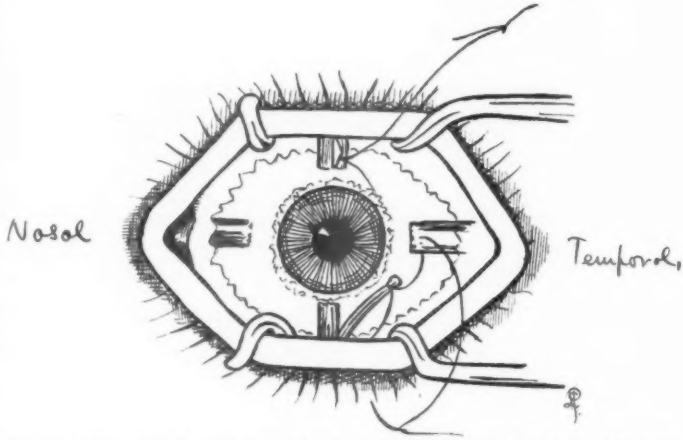

DIAGRAM II. Showing, above, ligature round fasciculus before detachment, and, below, needle with ligature passed under lateral insertion, but not tied.

Another point in the last case dealt with, although one achieved poise in the primary position, avoidance of diplopia and discomfort, and at least 20° of abduction, the abduction was much less than in the other two cases. This I believe was due to the fact that here I deliberately

slit the tendons up as far as I could, so that there was very little strain on the grafted portions. In the others there was considerable tension. It appears to me that that is probably an essential element in the success of the operation. The problem is possibly very largely a mechanical one. In this connexion I shall quote the latest reference to the operation of which I am aware. It occurs in the *American Journal of Ophthalmology* for November, 1934, from the pen of M. E. Marcove:

> A girl had a 25° convergent squint, due to paralysis of the lateral rectus of congenital origin. When she looked to the right she saw double and became nauseated.
>
> The medial halves of the superior and inferior recti were freed and attached underneath the insertion of the lateral rectus. A recession of the medial rectus was also done. The final result was complete relief of the convergent squint, and an ability to abduct the right eye about 25°. According to various authors, a rotation of 15° to 20° is considered a good result.

This operator does not say why he used the medial halves of the tendons for his grafts, but it may be that in America it has been

FIGURE IX. After operation, looking ahead.

FIGURE X. After operation, looking to left.

found in practice that a good degree of pull from the reinforcing tendons to their attachment is desirable; and it may be that this is a device which insures that there shall be this strain.

I believe that the amount of abduction obtained is probably a matter of technique. At the same time, that a sufficient amount of abductive power to free the patient from discomfort, and to keep the eyes parallel in the primary position, can be obtained without fear of failure has been demonstrated to my complete satisfaction.

ADDENDUM.

Since reading the above paper I have additional experience of three more cases. One, that of a man of fifty-nine years, with a left abducens palsy of two

FIGURE XI. Before operation, looking ahead.

FIGURE XII. After operation, looking ahead.

FIGURE XIII. Before operation, looking to left.

FIGURE XIV. After operation, showing amount of movement to left.

years' duration. He had distressing diplopia, a convergent squint, and carried his head sideways. I did the same operation, with a recession of the medial. The result was good, but, anxious to make it better, a fortnight later I again tenotomized the medial. The final result was more than 30% of abduction, and complete relief of all diplopia and discomfort. This is shown in Figures IX and X. A photograph of the original condition was not obtained.

Another case was that of a boy of two, with a left paralytic squint from birth. This was unequivocal, as, when he fixed with the squinting eye, the convergence of the good eye was very much greater than the original squint.

In this case, following the precedent of Marcove, mentioned earlier, and also the suggestion of my colleague, H. St. Vincent Welch, who has recently published a well reasoned article in *The Medical Journal of Australia* of September 7, 1935, on the mechanics of this operation, I used the medial third of the vertical tendons, bringing the detached slip behind the standing part. This is undoubtedly more effective mechanically, and also, as Welch points out, lessens the adductive power of the vertical muscles, thereby allowing the obliques to utilize their abductive functions unhampered.

I did a complete medial tenotomy (subconjunctival), but as, a week later, abduction was slightly impaired, I reapplied the medial slightly behind its original insertion.

The result is shown in the accompanying photographs (Figures XI to XIV).

The last case was that of a child of five, with a paralytic squint. Here I used the medial third of each vertical tendon, and a week later did a tenoplasty of the medial with three cuts. There was almost complete abduction, but a decided loss of adduction, which I shall have to correct by an advancement.

It appears to me that, if the medial parts of the vertical tendons are utilized, one must be very conservative in handling the medial rectus, so great is the abductive effect of this procedure. Whereas, if one uses the lateral portions, one can afford and needs to do a fairly free tenotomy of the medial tendon.

A nice discrimination is needed, and further experience will determine what is precisely the best technique.

In the six cases I have operated on I have had the best results in those in which I used the lateral parts of the vertical tendons in combination with some form of slackening of the medial rectus.

THE SUSPENSORY LIGAMENT OF THE PELVIC VISCERA.

By W. E. A. Hughes-Jones,
Melbourne.

[From the Department of Anatomy, University of Melbourne.]

The student of anatomy is to be pitied in his battle to understand the "fascia of the pelvis" and the supports of the pelvic viscera, so hopelessly involved is the minute description of "fascial attachments" which is given in the textbooks. Paterson,[1] Elliot Smith,[2] Derry[3] and Cameron[4] have shown the principles of pelvic fascial disposition with more clarity and their papers make the writer's description redundant in many respects. The arrangement of the visceral supports is simple, and this paper contains a description of the only ligament which matters inside the pelvis, both male and female, that is the "suspensory ligament" (Paterson) of the pelvic viscera, one portion of which has been especially termed the utero-sacral ligament. But it should be remembered that the great factor in the support of the pelvic viscera is the *levator ani* muscle. The suspensory ligament is a contributory factor concerned rather with maintenance of posture than with weight-bearing.

For the purposes of this study the pelvis was divided in the sagittal plane, the mucosa and musculature of the rectum were removed from each half and the fibrous adventitia of the rectum was carefully raised, the posterior portion of the suspensory ligament being exposed. The fore part of the ligament was studied in an oblique (horizontal) section made in the plane of the ligament.

The diagrams explain the disposition of the suspensory ligament much better than words. On each side the sacral attachment is to the fascia over the piriformis muscle over an area corresponding to the upper part of the greater sciatic notch (Figure I). This attachment corresponds to the mouth of a funnel into which enter the visceral branches of the anterior division of the hypogastric artery and the visceral branches of the third and fourth sacral nerves. This funnel rapidly narrows to a neck which is the hindmost part of the suspensory ligament.

The ligament then rapidly widens as it passes forwards and downwards (Figure II). The lower border is quite free, standing out about 1·25 centimetres (half an inch) from the surface of the ilio-coccygeus muscle. The whole ligament has no attachment other than its insertion to the *levator ani* muscle and its enveloping fibrous sheath. A finger

passed under its lower border can explore the whole of the pelvic surface of the *levator ani* and the parieties to the ilio-pectineal line and to the side of the bladder without encountering any structures joining the suspensory ligament to the pelvic floor. The exploring finger can readily draw the ligament inward and demonstrate its great strength.

A description of the further disposition of the ligament in the male follows. As the ligament approaches the postero-lateral angle of the bladder and the vesicles and prostate, it is in the form of a high strong band (Figure II) reaching below to the apex of the prostate and the triangular ligament and above to the postero-lateral angle of the bladder.

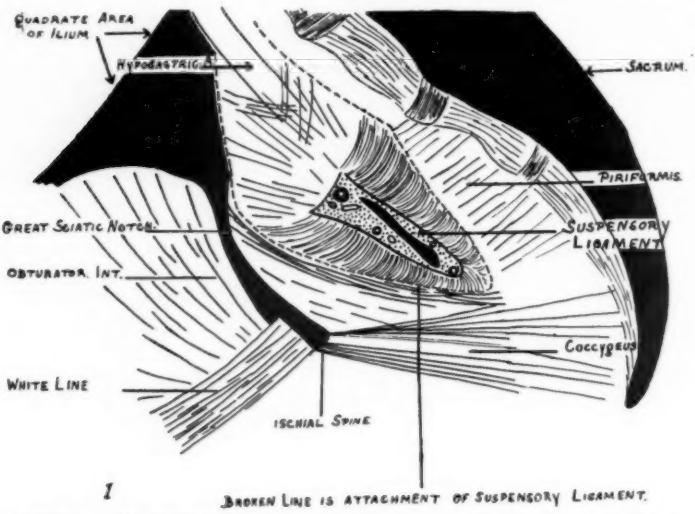

FIGURE I. The attachment of the right suspensory ligament in the female. For the interpretation of the structures shown in the ligament itself reference should be made to Figure VI.

The line diagram (Figure IV) illustrates the next stages of the description. A strong septum sweeps medially in front of the rectum and behind the prostate, and is continuous with its fellow of the opposite side. This is the structure described as the fascia of Denonvilliers, and it can be split into several layers, a circumstance not affecting its functional unity. Thus the ligaments coming down from the region of the greater sciatic notch form a strong sling which is attached to the back of the prostate and to the vesicles.

A further strong extension of the suspensory ligament on each side passes to the postero-lateral angles of the bladder and prostate (Figure IV), the vesicles lying between this extension and the fascia of Denonvilliers. This extension bears the blood vessels of the prostate and bladder.

The foremost expansion of the suspensory ligament in the male is well defined, passes forward round the lateral and anterior surface of the prostate (Figures IV and II) to join its fellow of the opposite side and to form a sling surrounding the prostate and anchoring it to the sacrum. A feeble upward continuation of this expansion passes to the side of the bladder. From the front of the sling around the prostate a strong set of fibres passes to the back of the pubis on

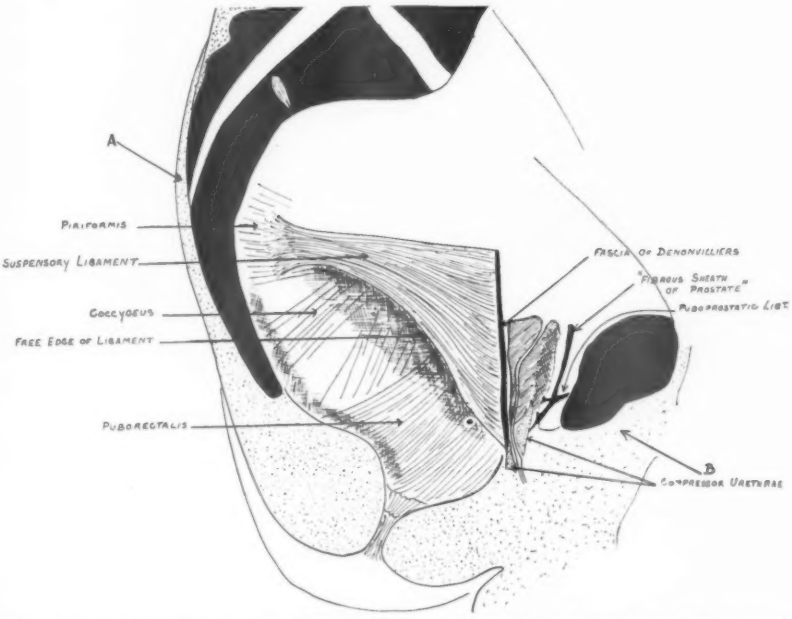

FIGURE II. Sagittal section of male pelvis, showing the suspensory ligament after removal of the rectum.

either side of the mid-line, surrounding the pubo-prostatic ligaments (Figure IV).

In short, the suspensory ligament in the male consists essentially of two slings, one behind and the other in front of the prostate, with a guy-rope to the pubis in front and an anchor to the postero-lateral borders of the bladder and prostate. The general plane of the ligament is very oblique and is ideally arranged to support the viscera from the parietes.

The suspensory ligament in the male has been described so that the fundamental similarity of the ligament in the female will be apparent. In the female there is the same discrete attachment of the posterior end of the ligament (Figure I) and the ligament possesses the same

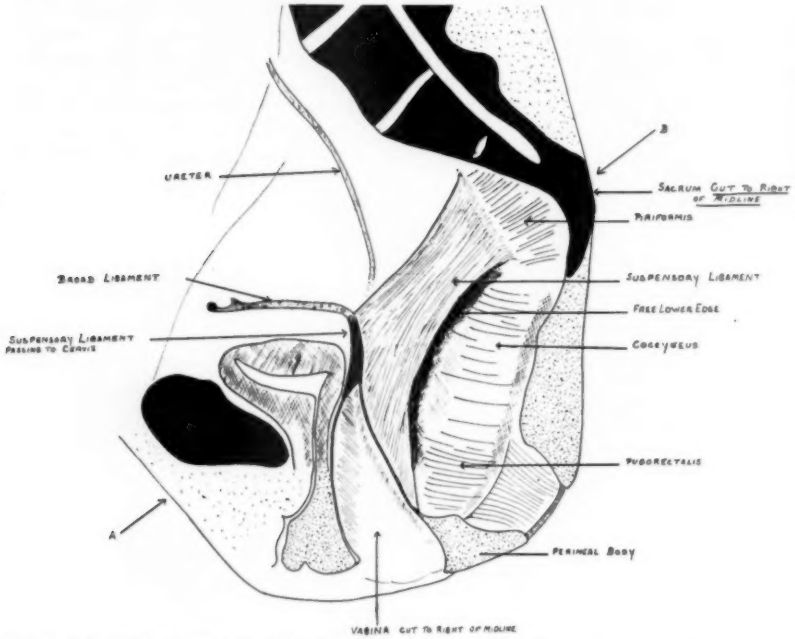

FIGURE III. Sagittal section of female pelvis, made to the right of the mid-line, showing the suspensory ligament after removal of the rectum.

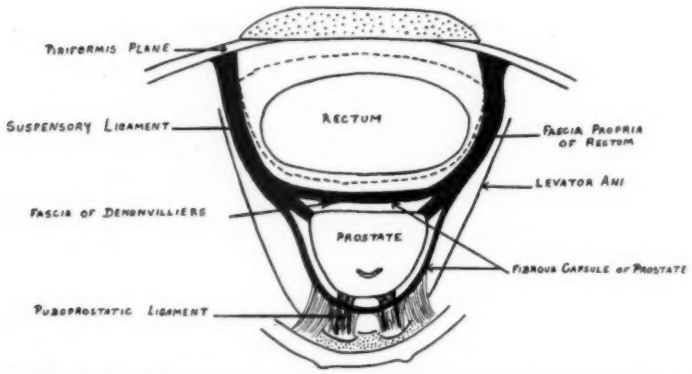

FIGURE IV. Arrangement of the suspensory ligament in the male. The section is made in the line A-B of Figure II.

freedom from any attachment to the inner surface of the *levator ani* and its fibrous tissue envelope (Figures III and V). The ligament can be drawn inward with the greatest ease, and this manœuvre demonstrates the strength of the attachments to the bladder, cervix and vagina. As the suspensory ligament approaches these viscera, it spreads into a very strong wide septum which extends vertically from the level of the isthmus of the uterus to that part of the vagina which passes between the *levatores ani* muscles (Figure III). The tissues of the medial surface of the ligament spread inwards as a particularly strong layer to be attached most firmly to the whole length of the lateral margin of the vagina, to the dome of the lateral fornix and to the lateral margin

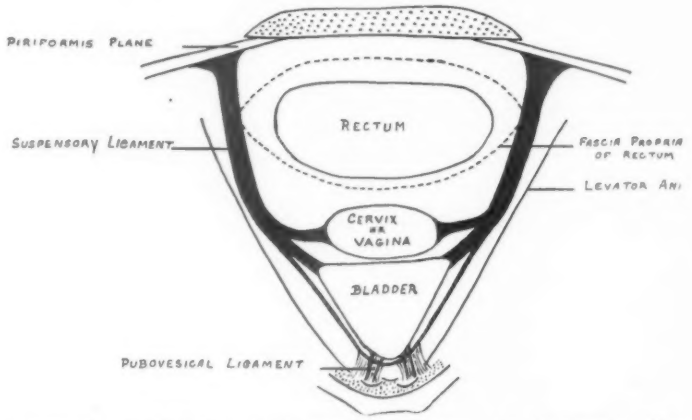

FIGURE V. Arrangement of the suspensory ligament in the female. The section is made in the line A-B of Figure III.

of the neck of the uterus as high as the isthmus (Figures III and V). Thus the vagina and the neck of the uterus are held firmly in a sling, the two extremities of which are attached to the parietes in the region of the greater sciatic notch (Figure V).

Another expansion of the suspensory ligament passes to the postero-lateral border of the bladder, is quite strong and bears the vesical blood vessels (Figure V). Whereas in the male a strong expansion passes around the front of the prostate, in the female the expansion which passes round the sides and front of the bladder is quite weak (Figure V). It is attached to the pubis on each side of the mid-line, covering the pubo-vesical ligaments.

The very oblique plane of the suspensory ligaments is such that the posture of the neck of the uterus and the vagina is maintained most efficiently.

Several extraneous matters can be added to this description of the supports of the pelvic viscera. The *fascia propria* of the rectum (Figures IV and V) has a linear attachment to the inner surface of the

suspensory ligament. The peritoneum of the utero-sacral folds is anchored to the upper border of the ligament. The inner part of the base of the broad ligament and the suspensory ligament converge as they approach the neck of the uterus, and in this angle the ureter lies close beneath the peritoneum.

The vesical and middle hæmorrhoidal arteries and veins enter the lower portion of the suspensory ligament to pass to and from the bladder, prostate and perirectal tissues (Figure VI). The uterine artery and veins run in the upper and front border of the ligament (Figure VI). When the finger is passed under the ligament as described earlier

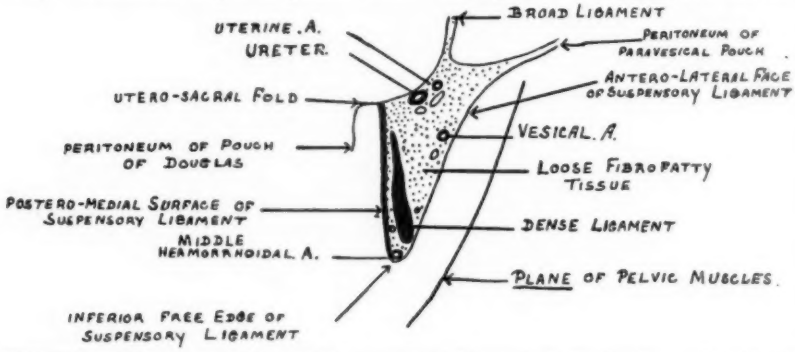

FIGURE VI. Transverse section of the suspensory ligament of the female, made midway between the posterior attachment of the ligament and the lateral margin of the cervix.

in this paper, these vessels are lifted upon the finger and their origin near the upper margin of the greater sciatic notch can be felt (Figure I).

A transverse section of the suspensory ligament is shown in Figure VI. A very strong band lies near the inner surface and around this fibro-fatty tissue, blood vessels and nerves are gathered, tissues which, weak in themselves, form a strong suspensory ligament when bound together.

SUMMARY.

One pair of ligaments is alone responsible for the maintenance of the posture of the pelvic viscera in the male and female. Paterson aptly named these the "suspensory ligaments". The ligament is laid down in the line of pull of the viscera, and through it the blood vessels, nerves and ureter reach the viscera.

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THROMBOCYTOPENIC PURPURA: A CLINICAL STUDY.

By F. Tidswell and R. B. Wade.

[From the Royal Alexandra Hospital for Children, Sydney.]

Thrombocytopenic purpura has presented itself at this hospital in two forms. In one it is an acute and usually severe condition with a high fatality; but if the patient survives there is no recurrence and recovery seems to be complete. In the second form also the onset is sudden, but the course is entirely different. The purpuric attacks are less severe, but recur at irregular intervals with intervening periods of comparative well-being, composing a chronic illness which may endure for months or years. These forms correspond to the two types usually described under the term *purpura hæmorrhagica*. The very severe cases of the acute form would come under the heading *purpura fulminans*. The recurrent form is now commonly referred to as essential thrombocytopenia, but has been given a variety of names by different authors.[1] Our available records concern 18 cases which have come under notice mostly during the past five years. Fourteen (seven occurring in males and seven in females) were of the acute and four (three in males and one in a female) were of the chronic type. The ages of the patients varied from sixteen months to thirteen years, eleven being under five years of age.

In the acute cases six of the patients died (approximately 43%) in three, four, six, eight, sixteen and thirty-nine days respectively after the first purpuric signs appeared. The eight surviving patients remained in hospital for from one to five weeks, during which time all purpuric signs had disappeared; and this we should regard as representing the usual periods in uncomplicated cases. In two of our patients vascular obstruction resulted in gangrene which necessitated treatment for five and six months respectively.

There were no definite preliminaries to the attack in these cases. The children had had the usual measles, pertussis, mumps *et cetera*, but not invariably; "sore throat", cold, bronchopneumonia and "feverishness" were too infrequent to be regarded as other than incidental. There is no reason to think these various conditions had any toxic or anaphylactoid or other predisposing influence; there is certainly no one such factor which stands in manifest causal relationship to the onset.

[1] *Morbus maculosus* (Werlhoff), essential thrombocytopenia (Frank), thrombolytic purpura (Kaznelson), hæmorrhagic thrombasthenia (Glanzmann), *purpura thrombopenica* (Schultz), hæmogenia (Weil) *et cetera*.

The hæmorrhages have taken the form of spots or bruises of varying degree, and were of irregular but more or less generalized distribution. Quite often blood has been vomited or passed with the urine or fæces. Often also there has been epistaxis (sometimes the first noted sign), oozing from the gums, and occasionally bleeding from the ears. These various events were equally present in the surviving and fatal cases, and so have no prognostic significance. No doubt visceral bleedings were also frequent; at all events they were present in the two cases which came to autopsy (lungs, liver, intestinal walls, heart, pericardium). In two of the fatal cases gangrene seemed to be threatening, and in two of the surviving cases actually occurred. Pressure on vessels and interference with blood supply were not so evident in these cases as was expected. In one of them, whilst the foot was the site of a moist gangrene such as might result from blockage of a large vein, there was no hæmorrhage into the tissues to account for it, and further gangrene of the fingers commenced exactly at the web, and was perfectly symmetrical in all four fingers affected (Figure I). In the other case there was a large gangrene of the left foot and leg (Figure II). It is not so easy to visualize these lesions as due to pressure, and the first case especially suggests the possible operation of some neuro-vascular mechanism. The wheals of the allergic purpuras may have a similar causation and be not due to endothelial damage as usually held. This idea arises from the apparent need for some further explanation of these events; it is merely

FIGURE I. Showing gangrene perfectly symmetrical in all four fingers affected.

FIGURE II. Showing large gangrene of the left foot and leg.

speculative, but is offered as being worthy of some consideration. The spleen is recorded as enlarged in two cases—one fatal, one not: it was doubtful whether it was enlarged in two others; it was not enlarged in four cases, and the condition is not recorded in the remaining cases. It follows that the spleen is not usually enlarged in this condition. Enlargement of the liver is noted in two cases, both fatal. There is no record of enlarged lymph nodes.

Reference to the blood counts generally is made elsewhere; place is given here for the thrombocytopenia as being one of the clinical diagnostic features of the disease. In 11 of the cases there was definite thrombocytopenia, the initial counts being 47,000, 81,000, 15,000, 50,000, 83,000, 79,000, 60,000 and 100,000 per cubic millimetre respectively; "too few to count" and "none" were two of the records. In another case the initial report was "platelets abundant", but a count five days later gave 89,500 per cubic millimetre. In another case the initial count of 206,000 was vitiated by previous transfusions; a month later there were 60,000 platelets per cubic millimetre. In the remaining case no count was made.

The subsequent counts are less representative as being influenced by treatment. Recovering patients showed increase of platelets, not necessarily progressive, but a general advance, despite some remissions. Conversely, in fatal cases low counts were retained. In one of these no platelets at all were seen in any of the four counts made during twenty-six days. In another the count rose from 89,000 to 293,760 during the week prior to death, which seemed to have been precipitated by violent gastric hæmorrhage in a patient whose condition appeared to be improving. The platelets continue subsequently on the safe side in the patients who have recovered; as far as we are aware none of our eight patients has relapsed. Four of them are out of present reach, but in the other four the platelets per cubic millimetre recently numbered 220,000 (seven years), 180,000 (three and a half years), 120,000 (one year) and 160,000 (seven months), the times having reference to the periods elapsing since the last signs of purpura. Two of these children have had amputation and repair operations, and one of them has also had tonsillectomy, another had tonsillectomy and four teeth extracted, without excessive bleeding in any instance. From these issues it appears that, although there may be some shortage of platelets, there is no manifest defect of health, and that once the attack is over it is over for good and all.

In all four of the chronic cases there was a history of repeated bleeding, for several years in one instance, for two years in another, and for one year in the other two cases. The form of the bleedings was the same as in the acute cases—generally distributed ecchymoses, epistaxis, oozing from gums, hæmatemesis, melæna and hæmaturia, without apparent cause. The interval between attacks was variable, and even in these quiescent periods trauma produced disproportionate bruising. In one of our cases petechiæ appeared on the limb below a ligature.

In two of these cases the spleen was enlarged; in the other two it was not palpable. There is no recorded enlargement of the liver. In three of the cases there was manifest thrombocytopenia; in the fourth there were 110,000 platelets per cubic millimetre at the one count which we were able to make during the brief period the child was under our observation, and we have no further information about him. Of the three patients whose condition was better studied, two came to splenectomy, the third, still under observation, has not yet appeared to require this operation.

In our first splenectomy the blood platelets were remarkably variable prior to operation, being none, 6,580, few, 59,520, 60,172, none, 30,000, none, none, none, and a few per cubic millimetre in successive counts. After the operation the counts were 186,000, 171,426, 129,000 and 180,000 during the subsequent three months.

In the second case[1] the pre-operation counts were none, none and a few during three months; after operation the platelets rose to 104,300, were 131,000 a year later, and 99,000 a year later still.

Both of these patients are reported to be in good health, but we have no recent platelet counts.

Our third chronic case has been under observation for about fifteen months.

During this period the child has had several purpuric attacks consisting of spots and bruises occurring on the body, limbs, lips, soft palate and fauces, and she has had epistaxis and hæmaturia. The abdomen is soft, and the spleen is not enlarged. Her platelet numbers are very irregular. They have been below 100,000 per cubic millimetre in twenty of the twenty-three counts made (as low as 10,000), the remaining numbers being 110,000, 160,000 and 290,000. The initial count was 50,000, and at the present time usually ranges round 80,000, although the most recent is but 5,500 during a severe epistaxis.

In very obvious contrast to the patients whose spleens have been removed, this particular patient is not making satisfactory progress. The question of splenectomy is difficult by reason of the non-enlargement of the spleen and the general not serious condition of the child; but in any case the operation has so far been opposed by the parents.

The foregoing accounts are in accord with the usual descriptions of *purpura hæmorrhagica*, and well display the thrombocytopenia characteristic of the condition. The point of essential interest is the relationship of this feature to the purpura. For they are not invariably linked; thrombocytopenia may be lacking in the anaphylactoid purpuras. It is an obvious possibility that in the type under review the platelet deficiency may be no more than an accompanying sign of some other condition which is the real cause of the abnormal bleeding. Such a condition might be one influencing the blood itself (that is, its coagulation), or one which prejudices the integrity of the vascular wall—factors which have both been invoked in explanation of hæmorrhagic states.

[1] The operation in this case was performed by Dr. G. A. Hardwicke, to whose courtesy we are indebted for the publication of the counts made by us.

It is not easy to secure a basis for valid consideration of the blood aspect of the question. The coagulation of blood is paradoxically at once well known and unknown. The fact is a commonplace, its essence a mystery. Of several theories none is wholly acceptable, and none wholly comprehended even by experts. In a general way we understand that soluble fibrinogen and thrombin interact to form solid visible fibrin, the threads of which entangle the remaining blood and corpuscles, forming a coagulum. The subsequent contraction of the fibrin squeezes out fluid with the ultimate issue of clot and serum.

Fibrinogen exists in the circulating blood to which it is supplied by the liver (suggested as formed from the globin of hæmoglobin after the hæmatin is removed to form bilirubin). Modern ideas are to the effect that fibrinogen does not become fibrin within the vessels normally because it is not free there, but is united with albumin and globulin to form a protein complex. It becomes vulnerable only when the aggregate is broken up and it is set free. This happens when the physical, electrical, chemical or other factors governing colloid conditions are disturbed, as by contact with alien matter within the vessels or when the blood is shed.

But in any case fibrinogen does not by itself become fibrin; for this it has to be acted upon by thrombin. Thrombin does not exist in the circulating blood, but is there represented by a precursor—prothrombin. The source of prothrombin is unknown (possibly it is the liver), but, like fibrinogen, it is regarded as part of the protein aggregate. It becomes converted into thrombin in the presence of calcium when acted upon by cephalin. The production of thrombin is thus complex. Calcium is present in the plasma and is always available. Cephalin (thrombokinase) is not ready formed, but it exists as a phospholipoid constituent of all cells—leucocytes and platelets as well as tissue cells—and is liberated when these are disintegrated—promptly from platelets in shed blood. The cephalin is supposed to render the prothrombin susceptible to the calcium, but the exact mode of action is unknown.

Fibrin is usually thready, but under certain conditions may be a structureless gel. Its contractility is associated with the threads; in its gel condition it is non-contractile. The formation of threads seems to be conditioned by the presence of platelets (cephalin); non-contractile clots are usual in platelet deficiencies.

The normal intravascular fluidity of blood is thus regarded as due to the stabilization of fibrinogen and thrombin by their aggregation with other plasma proteins. The idea is that the unshed plasma is not a mixture of several proteins, as formerly taught, but a coordinated colloid complex in which the unstable fibrinogen and thrombin are held intact by the more stable albumin and globulin. These various fractions appear as separate entities only when the complex disintegrates. The bond is strong enough to resist small intravascular influences such as

must constantly be exerted, but is overcome by greater disturbances such as occur on shedding.

According to some authorities, intravascular fluidity is due to the existence of an antiprothrombin which prevents any prothrombin liberated being converted into thrombin. Such a substance—heparin from its suggested liver origin—has been found in blood. When blood is shed the heparin is neutralized by cephalin and the prothrombin is free to become thrombin under the action of calcium. According to this view, fluidity after shedding might depend on excess or non-neutralization of the antithrombin (heparin), but this would be relative to the amount of cephalin (platelets), which is thus also important in this theory.

Whilst we must claim some indulgence for this endeavour to get a lucid view of coagulation, we feel that at least it defines the position of the platelet in that process, and that is the point of present concern with regard to our cases. It is clear that there may be a lack of cephalin due to deficient platelets, and so imperfect coagulation. Our coagulation times (recorded in fourteen cases) were nine times between the normal (three and six minutes); in the other cases they were short twice (one and a half and two and a half minutes) and long three times (nine, nine and a half and ten minutes). The coagulation times are thus usually within the normal limits. We have records of times of seven and a half minutes in non-purpuric cases by the same method, so that the few longer times in our purpuras are not specially significant beyond suggesting some disturbance. The bleeding times in our cases were within the normal two to four minutes in four cases, below it in two, and above it in seven cases, being in these six and three-quarter, seven, seven, seven, twelve, twelve and twenty minutes respectively. We have no comparable experiences in our few non-purpuric cases, and so conclude that our figures accord with the current view that the bleeding time is usually prolonged in *purpura hæmorrhagica*. Our observations on clot retraction are too few to afford valid inferences. The irregularities shown are suggestive of coincidental rather than causative clotting defects. The usually normal coagulation time associated with a prolonged bleeding time is more indicative of a vascular lesion than of defective functioning of platelets.

The blood platelets have been known for more than half a century, and in recent years have become regularly noted in blood examinations. As seen in ordinary stained blood smears, they look like fragments of cells. They vary in size from one to three micromillimetres, a red corpuscle being seven micromillimetres. They tend to adhere to one another and to disintegrate in the shed blood, qualities which explain their function and at the same time impose some difficulties in their preservation and enumeration.

Their source has been variously ascribed to the (i) red corpuscles, (ii) white corpuscles, (iii) plasma (precipitate); but nowadays they are not considered as derived from any circulating element, but to be formed

in the marrow. They are diminished in marrow depression, for example, aplastic anæmia, and increased in marrow exaltation, for example, myeloid hyperplasia after hæmorrhage. They are commonly described as being broken off the pseudopodia of megakaryocytes projected into the blood sinuses of the marrow. This accidental sort of business does not account for their constancy in presence and numbers, and the most modern idea is that they are derived from a special formative marrow cell which is under physiological regulation and which is denucleated and fragmented before leaving the marrow. This process is comparable to that by which a non-nucleated red corpuscle is derived from a nucleated marrow erythroblast, and the close serological relationship between platelets and red corpuscles permits the conception of kinship and the possibility of a similar developmental career. In the blood of birds the platelet is represented as evolutionary precursor by a complete nucleated cell—thrombocyte, a name now applied also to platelets. No such cell has been seen in mammalian blood, normal or diseased, nor in that of the youngest embryos examined; platelets take its place. By using platelets as antigen there has been produced an antiplatelet serum which contains a specific antibody for platelets (Bedson).

Certain poisons—pyridine, toluenediamine *et cetera*—affect only platelets. In blood their numbers fluctuate independently of the other cells. Thus there is much evidence in favour of their separate individuality. But whether from megakaryocytes, as described by Homer Wright, or from a special mother cell as the later idea has it, there is general agreement that platelets are born in the marrow; understandably, therefore, their deficiency has been ascribed to marrow defect —the thrombasthenic aspect.

Purpura is accompanied by anæmia, usually regarded as secondary to the bleeding. Our records show that it was not extreme in acute cases in which recovery occurred, the figures being, respectively, 3·7, 4·0, 3·1, 3·8, 4·2, 3·4, 2·1 and 2·9 million of red corpuscles. In the two last cases a rapid advance to over three million occurred. The hæmoglobin was correspondingly reduced, so that the colour indices were not unduly low—0·8, 0·76, 0·6, 0·8, 0·6, 0·89, 0·71 and 1·0 in the several cases. There was no anisocytosis, nor poikilocytosis, few nucleated red cells and only mild polychromasia.

In the fatal cases the anæmia was generally more marked, 2·7, 4·3 (4·1), 1·8, 2·0 and 3·5 (2·1) million being recorded (figures in parentheses being subsequent counts). In the second case the end came with quite high erythrocyte figures. The hæmoglobin was low (40%) in this second case, and in one other case it was 30%; the colour indices were 0·49 and 0·5, but in the other three they were 1·1, 0·8 and 0·9 respectively. Here again there were no structural defects of the red corpuscles, only occasional normoblasts and some polychromasia.

There was no approach in any of these cases to an aplastic state of the marrow. The white blood cells were invariably within the normal limits, varietally correct for the various ages, and never accompanied by

abnormal or immature cells. Thus there was no evidence of any malcondition of the marrow sufficient to throw blood formation generally off its accustomed path, and inferentially none that would have disturbed thrombopoiesis. Certainly a specially selective agency is not eliminated, but on the other hand is not indicated by any circumstance of the cases. In this connexion it may be recalled that antiplatelet serum caused purpura (Ledingham) which was not produced by anti-red nor anti-white cell sera (Ledingham and Bedson), and that antiplatelet serum, although agglutinating both platelets and red corpuscles, contains a specific antibody for platelets (Bedson). Manifestly there may be in purpura some noxious agent the activity of which is restricted to platelets, but this is not vouched for by any facts within our knowledge.

Blood platelets occur in the circulation in numbers ranging between 250,000 and 400,000 per cubic millimetre, but there is a wide range in health, and still wider variation in disease. In purpura the numbers may be very low. In our cases they have been sometimes so few that none were to be found in the usual preparations. There seems to be no exact quantitative relationship between their numbers and the bleeding. The danger zone is given as 50,000, but bleeding occurs with higher numbers (70,000 to 80,000 in our case), and may not occur with fewer platelets (not observed in our cases).

From the time of their first discovery, platelets have been associated with coagulation, and nowadays are regarded as the suppliers of cephalin as already mentioned. Their rôle in abnormal bleeding seems to be limited to hæmorrhagic purpura regularly, and to anaphylactoid forms occasionally. They may be diminished in pernicious anæmia, leuchæmia, and other marrow stresses in which bleeding occurs, but are usually normal in other and more definite hæmorrhagic disorders, for example, hæmophilia and hæmorrhage in the new born. This singularity of their varying relationships to bleeding diseases, in contrast to their regular presence in considerably and fairly defined numbers during health, suggests that they are designed to perform some more usual work than the clotting of blood in gross hæmorrhage, which may seldom or never occur. It has been surmised that their constant job is protection of the vessel walls, and the sealing of small lesions which may be supposed to be of frequent occurrence for one reason or another. Some authorities aver that it is when these lesions are very widespread (for example, fatty degeneration of the endothelium) and so many platelets are being used as plugs, that shortage (thrombocytopenia) results; the sealing becomes incomplete and petechial hæmorrhages (purpura) occur. The mechanism of hæmorrhage in *purpura hæmorrhagica* is petechial—diapedesic migrations of corpuscles and plasma through the apparently intact vessel walls in contrast to the massive hæmorrhage through wounds as in hæmophilia. But the integrity of the vessel walls may be merely apparent. It is held by some that the same influence which destroys the platelets also injures the vascular endothelium. The blood then finds issue through the weak spots not now properly sealed. Affinity

between endothelium and platelets which would render them susceptible to the same noxa seems possible, but we are not aware of any evidence of a relationship between these two closer than between them and the other blood corpuscles. They all have a common origin, and might be expected to have a more or less common suffering. But, as already shown, this is not the case in purpura.

The belief that platelets normally end their life in the spleen adds another cloud to issues already too obscure. The spleen is a mysterious organ. In the embryo it makes all blood cells. In the post-natal life it continues to form lymphocytes, but has abandoned the other blood cells to the marrow in which their production is concentrated. But the spleen may reassume blood formation generally under stress—myeloid metaplasia. It normally maintains its relationship to blood formation in a negative way by destroying the red corpuscles (and platelets), and it is asserted that it exercises a regulating influence over the marrow process. By destroying red cells *et cetera*, and by acting as a reservoir of blood (one-fourth of the red corpuscles are said to be normally parked in the spleen), it determines the number of red cells in the circulation, and can compensate rapidly in anoxæmic conditions and perhaps more slowly to balance less urgent requirements. It is held by some that in the case of platelets also it acts at once as a reservoir and a grave—the thrombolytic aspect. The chief support of this idea is the increase of platelets after extirpation of the spleen, which may lead temporarily at least to even excessive numbers. It occurred in our two spenectomies, though not to undue degree. The particular constituent of the spleen which destroys the platelets is the macrophage tissue, which also destroys the red corpuscles. Shortage of platelets is held to be due to excessive activity of this reticulo-endothelium. The post-operative increase is due to removal of this large mass of platelet-eating cells. Since the spleen is just one of the principal, and not the only, depôts of this tissue, benefit, though usual, is not inevitable even in typical cases, and in cases in which splenectomy fails, other reticulo-endothelial areas are involved and keep up the destruction. It is to be supposed that from this point of view these other areas are equally or more responsible when the spleen is not enlarged, as would apply in most of our acute, and in one of our chronic, cases. It has been reported that in acute cases the patients are not only not benefited, but are usually prejudiced by splenectomy, so that the acute condition cannot be due to hypersplenism, or not to it alone. It would seem that real benefit from splenectomy can be expected only in the more chronic cases in which splenic enlargement offers some assurance that this particular reticulo-endothelial mass is the chief, if not the only, one concerned. It is clear that the ultimate issue must depend on whether the extrasplenic tissue remains quiescent or develops a compensatory voracity for platelets which nullifies the loss of the splenic group. But even if macrophagic destruction be accepted as the explanation of the thrombocytopenia, there still remains the reason for the reticulo-

endothelial gluttony for platelets. The disturber of the regulating mechanism is most easily conceived to be damaged or effete platelets needing more vigorous elimination than is called for when these elements are healthy and intact. Morphological variations of the platelets interpreted in this sense have been reported. But there is still the further issue that reduction of platelets alone will not cause purpura. Anti-platelet serum will, but "agar-serum" will not, produce purpura, though both reduce platelets (Bedson). This fact has been regarded by both Ledingham and Bedson as indicating the operation of some factor in addition to platelet destruction, and which they think is damage to the vessel wall. It is a common belief with regard to *purpura hæmorrhagica* that platelets and vascular epithelium are simultaneously injured. Bedson has described swelling of the endothelium and its projection into the lumen as possibly indicating vascular change. Such swollen projecting endothelium was quite apparent in the vessels of lymphatic nodes of one of our *post mortem* cases from which we have histo-pathological material—unfortunately scanty and collected before we were alive to the possibilities. In these nodes and in an excised spleen there appears to be some excitement of the reticulo-endothelium leading to an unusual abundance of these cells. But neither condition is peculiar to purpura and in the very mobile organs of children may be no more than the expression of youthful activity and growth. In our sections we could see no other changes which had any bearing on the condition under review.

If then we assume, as seems necessary, lesions of both platelets and vascular endothelium, we have conditions ready for, but not necessarily causative of, purpura, and possibly this is as far as the facts can carry us at present. But in the depths of our minds we have the idea that the sudden appearance of widespread petechiæ is precipitated by vaso-spasm. This, demanded by the threatened or actual bleeding, fails to control it because of weak walls and deficient platelets; and by reason of the increased intravascular pressure produces the petechial extravasations of blood, just as the ligature does; and in certain areas so blocks the circulation as to cause thrombosis and gangrene. As already stated, some such event seemed to be in operation in the very symmetrical gangrene of the hand in one of our cases, and which is shown in the accompanying illustration (Figure I). These considerations seem to us to complete the clinical picture, and to afford the most feasible explanation of our experiences.

We do not derive much comfort from these conclusions: they do not afford much useful guidance for treatment. We think that in acute cases the patients really live or die according to the severity of the initial attack. Blood transfusion should be done as a routine measure, and perhaps it is apt to cause the balance to swing towards recovery in some cases; but it is hard to be definite on this point. Anyway, should the patients get over the acute condition, and get rid of

gangrenous material that may be there, their recovery will be final. In some chronic cases the patients would seem to be able to go through life with occasional epistaxis and other hæmorrhages, and they are left with only a slight secondary anæmia, and really no great harm done. There is no call for any drastic treatment in these cases. In others, when the condition occasions chronic ill-health and dangerous anæmia, splenectomy gives excellent results.

THE SURGERY OF THE LENS.

By E. A. Brearley,
Sydney.

The subject of this paper, the surgery of the lens, has been chosen because to my mind it represents the most important, and, at times, the most difficult surgical aspect of our specialty.

Now that ophthalmic surgeons are practising in every important centre in the country, expert attention is readily available, and is availed of at a much earlier stage in the course of trachoma, squint, senile ectropion *et cetera*, with the result that the number of cases requiring operation in these conditions is greatly reduced.

The normal lens is practically transparent, with a slightly yellow tint even in early life. This yellow tint increases with age, producing a yellowish or greyish appearance on oblique illumination, which often simulates cataract, and of which we should always remember to remind our students.

With the general increase of protein content which goes on until puberty, the insoluble proteins gain ground at the expense of the soluble proteins. The lipoid substances, especially cholesterin, increase in amount with advancing years. Nutrition is maintained by diffusion through the capsule, particularly through the posterior capsule. It has also been established that the lens has an internal oxidation system.

As a physiological process, the lens structure becomes more dense with age, and changes occur in the protein content. When coagulation of these proteins occurs for any reason, an organic opacity is found in the lens, and a cataract has now appeared. Such a coagulation may be brought about by radiant energy, by direct action or by interference with the normal oxidation system; but apart from such factors there seems no doubt that the mechanism of cataract formation is, in the main, obscure, and is analogous to the unexplained degenerative changes which occur in any specialized organ when it begins to approach the end of its, or the individual's, physiological life.

It is well known that the formation of opacities proceeds for some time, and then slows down, and sometimes apparently ceases; but on the whole the opacities increase in size and number, and eventually interfere with vision to such an extent that they pass from the class of scientific, to practical cataract.

Once there is sufficient disability to interfere with the individual's activities, relief should be available forthwith, irrespective of the degree

of the supposed maturity. Duke Elder has summed up the medical treatment of cataract very justly:

> It is true that in some early cases of acute cataract (true diabetic and traumatic), when the process has not progressed to actual coagulation of the proteins of the lens, transparency may be recovered by controlling the exciting conditions. It is also true that in all cataracts, variations appear to occur, due to changes in the fluid traffic and alterations in the refraction of the unstable lens; but it is as certainly true that in all cases an organic opacity, once formed, is immutable.
>
> Coagulation of protein is an irreversible chemical change. It seems not unreasonable to hope that in the future, when our knowledge of this change and the factors causing it have been more fully developed, it may be possible to prevent its incidence or delay its occurrence, but in the present state of our knowledge, while it is reasonable to attack and eliminate any exciting cause that is known or suspected, the only legitimate method of treatment is operative removal of the lens.

Until the epoch making work of Daviel in 1750, couching of cataract was the only operative treatment used, and it is interesting to note that for the next hundred years couching held equal favour with extraction amongst European surgeons, until it was finally abandoned in the middle of the nineteenth century.

Daviel's story is interesting. He was a French army surgeon, born in Normandy in 1696, and at the age of thirty-two had devoted himself exclusively to diseases of the eye. It was not until he was fifty-four that he determined to adopt the operation, extraction of cataract, in all cases, having taken five years to make up his mind.

Two years later he published his celebrated paper, reporting two hundred operations by the new method.

The patient sat in a chair, the upper lid being raised by an assistant, the lower lid being controlled by the surgeon. An opening was made into the anterior chamber with an instrument like a keratome. This was enlarged with a blunt pointed knife, and with scissors until only the upper third of the corneal margin was left intact.

The corneal flap was lifted with a small spoon, and the lens was incised with a sharp needle. Pressure was made through the lid on the lower portion of the wound, and the lens was expressed from the capsule. If prolapse of the iris occurred it was pushed back. It is interesting to note that in his first four or five cases he removed the lens entire with a spoon.

In one of his letters Daviel states that of his first two hundred and six operations, one hundred and eighty-two were successful.

The next important modification was that of Sharp, of London, who in 1753 adopted a single knife for the incision instead of the keratome, and who expelled the lens in its capsule. This, with modification, became the permanent instrument for the incision, which continued to be made below.

In 1801 Himly first used mydriasis as a preliminary to the extraction of cataract. In 1864 von Mooren advocated preliminary iridectomy.

In 1866 von Graefe introduced his linear incision in the sclera, close to the iris, performed with the Graefe knife, and included iridectomy as part of the procedure.

The corneal incision, originally below, and later to one side, or even horizontally across the centre of the cornea (Kuchler), moved permanently to the upper portion of the cornea about 1865.

The modern methods of cataract extraction fall under two heads: intracapsular and extracapsular extraction.

Intracapsular extraction is performed by the Smith, Barraquer, Knapp, or Sinclair technique. The last three methods strove to overcome the disadvantage of the Smith operation, in which the lens is dislocated and expressed by external pressure alone. All methods in the hands of their particular exponents yield good results, but whatever the reasons may be, there is no doubt that none of these operations has up till now threatened to replace intracapsular extraction.

In a paper by Foster and Benson read before the Ophthalmological Society of the United Kingdom last year, extracapsular extraction was stated to be the operation of election in 95% of cases in England.

One of the reasons which led to the adoption of extraction of the lens in its capsule (apart from special conditions applying to Indian clinics), was a belief that immature cataracts were inoperable by Daviel's method. It was held that in such cases an undue amount of cortex was left behind, constituting an immediate danger of iritis, and an ultimate complication of a dense capsular membrane. Smith on this point is very emphatic, and states that: "Extraction of immature cataract of the capsulotomy method is hardly justifiable." His reasons are: "A considerable amount of lens matter is left behind which swells up and becomes opaque, and is exceedingly likely to give rise to a severe form of iritis."

Treacher Collins put the position very fairly:

> Another fallacy to which at one time considerable credence was given, and which is still sometimes referred to, is that soft lens matter in contact with the iris excites iritis. That asepsic lens matter may lie in contact with the iris without any inflammation resulting we see frequently after needling operation for soft cataract. It has been shown that the aqueous humour alone is a bad medium for the growth of bacteria. If, however, a mixture of lens substance with the aqueous takes place, a medium is formed which is very favourable to their growth.

Another advantage of the intracapsule operation is that no capsular membrane is left behind, requiring a subsequent needling, an operation which I believe should be performed as a routine measure, if the best and most permanent results are to be obtained from the extracapsular method.

The grave disadvantage is that by destroying the diaphragm formed by the suspensory ligament and the posterior capsule, less pressure is required to produce vitreous loss.

I should like here to say a few words about the methods of classifying cataract as immature. It seems that lenses permitting partial

red reflexes, or with an iris shadow on oblique illumination, are dubbed immature, and a large amount of cortex is expected to remain after the extracapsular operation. Recent work with the slit-lamp suggests that our present standards of maturity and the amount of soft cortex remaining will require considerable modification. Bucklens states that: "Lenses with posterior capsular opacity and a large nucleus leave practically no cortex, though they may be relatively transparent." In fact, operations performed without distinction, on so-called mature and immature cataracts, have resulted in the opinion that one is not justified in the assumption that more cortex will remain in one case than another. I believe the day is not far distant when the amount that will be left may be accurately predicted in all cases. The absorption of cortex, therefore, is the important factor in convalescence, after the extracapsular operation.

There seems little doubt that any infection which complicates cases of extraction of cataract is generally introduced from without, either at the time of operation, or through the corneal wound, when union is prevented by the presence of tags of capsule.

As our operative field cannot be rendered aseptic, it follows that every entry of an instrument into the eye exposes the eye to risk of infection. The operator should, therefore, aim at reducing his manipulations to the minimum. Delay in closing of the wound by tags of capsule is a real complication, and this can be entirely avoided if the lens is expressed through the intact pupil, and if any iridectomy is performed after the iris has returned to the anterior chamber, which it generally does of its own accord. The extra resistance to the expression of the lens is negligible. Preliminary iridectomy I believe to require definite indications and not to represent the ideal procedure.

The ideal operation to my mind was that of Trousseau, of Paris. He used one instrument only, the Graefe knife, and with it he made his corneal section, pricking the capsule as he passed the knife across the anterior chamber. After expressing the lens through the intact pupil, he massaged the iris back into position, thereby insuring that all capsule was returned into the eye. How many of these simple extractions called for iridectomies next morning I cannot say, but even so, at the worst they had two entries into the eye, and at best only one. Contrast this with section, capsulotomy, iridectomy, irrigation, reposition of the pillars of the iris, and attempts to replace the capsular tags—six entrances in all.

While each surgeon ultimately adopts his own technique, and is guided therein by the results obtained, to those who may be interested, the following procedures may be discussed:

1. Corneal section and capsulotomy in one manœuvre in the manner of Trousseau. It is not necessary to rotate the knife, thereby losing aqueous, but merely to depress it at the near papillary margin, pushing straight on to clear the other margin of the pupil. It is very seldom

that the capsule does not readily tear under the pressure of expression. In such few cases capsulotomy has to be performed as usual.

2. Conjunctival bridge instead of flap. This is a great comfort in prominent eyes with restive patients. The extreme end of the bridge should be very narrow.

3. Expression of the lens through the intact pupil and under the bridge requires no extra effort, especially if the upper portion of the lens is cocked up by pressure just at or within the limbus below.

4. Late peripheral, or total iridectomy, according to the position taken up by the iris, after its return from under the conjunctival bridge.

If all goes well, as it does in nearly all cases, the eye has been entered twice, namely by the Graefe knife and the iris forceps. This procedure needs no variation in so-called immature cataracts.

May I, in conclusion, express the hope that the term "immature cataract" may be for ever banned. It has led to much misapprehension in the past, resulted in much ill-judged advice, and has caused countless numbers to wait, perhaps for years, for the relief which should have been forthcoming at once.

THE INTRACAPSULAR EXTRACTION OF CATARACT WITH SINCLAIR'S FORCEPS.

By A. L. Tostevin,
Honorary Ophthalmologist, Adelaide Hospital.

I am very mindful of my presumption in discussing the subject of the intracapsular extraction of cataract with Sinclair's forceps, when it has been so very ably presented to us by Sinclair himself in his presidential address to the Ophthalmological Society of the United Kingdom in May, 1932. I can only claim for my excuse the desire to impress upon those of you who have not already exploited his methods, the very decided service he has rendered to ophthalmic surgeons in evolving his instruments and technique for this operation. I feel confident that it will eventually become the method of choice by all who take the trouble to master the technique and to use it judiciously in suitable cases. There is no doubt that the intracapsular method of extraction has enabled us to remove immature cataracts, the removal of which could not otherwise be attempted.

The poor results obtained by some of the surgeons who have done intracapsular extractions by the different methods have no doubt been due to two things: (i) improper selection of cases; (ii) an inferiority complex brought about by the fear of loss of vitreous, which not only interferes with the surgeon's precision, but also makes itself felt in some subtle way by the patient, who in turn does not cooperate to the operator's advantage.

I shall endeavour to show you the results of my experiences in regard to the first and hope that you will be able to profit by them. As for the second, if any man really fears the operation it would be better if he did not attempt it, because he will certainly get poor results.

Part of the object of this paper, therefore, will be to give you confidence by demonstrating my results and from the failures I have had to help you to decide upon which cases to avoid for this operation.

I shall also show how I endeavour to get the best cooperative effort from my patient, which is a very great help. Early in 1930 I first began to use the old type of Sinclair's forceps, which I exhibit today. To gain confidence and to get accustomed to the forceps, I began by doing intentional capsulectomy, that is, after picking up the lens capsule, I put the point of a Ziegler's needle into the periphery of the lens and then withdrew the forceps, which brought with it a piece of capsule from the pupillary area. I then proceeded to an ordinary expression of the lens. I found the result of this procedure very satisfactory, inasmuch

as absorption of any lens remains seemed quicker from the more ready access to the aqueous, and needlings were seldom necessary because of the absence of an anterior capsular layer in the pupillary area. After doing fifteen capsulectomies, I felt confident to proceed to the next stage and endeavoured to deliver the lens in its capsule. My first three attempts ended in unintentional capsulectomy, that is, the capsule broke and the forceps came away with portion of the capsule, so I simply proceeded to ordinary expression. I then made a successful attempt and thereafter my unintentional capsulectomies became less frequent. The thrill of successful termination and the ultimate disclosure of its very decided advantages led me to exploit the operation further, and at the beginning of 1932 I acquired the new type of Sinclair's forceps and found them a decided improvement, which helped my results considerably. I have now performed 65 intracapsular extractions, 48 of which terminated without any complications and obtained vision from $^{6}/_{6}$ to $^{6}/_{12}$.

Five patients had partial prolapse of iris at the first dressing, but these were in the early days of my efforts. In four cases I experienced loss of vitreous, but this occurred in cases in which today I should not attempt the intracapsular operation.

As soon as I have satisfied myself that the patient needs a cataract extraction, my first endeavour is to give him confidence. The fear of a cataract operation is still very prevalent amongst the public. They associate it with tortures that have to be endured by lying flat on the back with sand bags holding the head as if in a vice. They fear the actual operation, and because of no general anæsthetic they imagine it necessarily painful. All this must be dispelled in your consulting room. Allow the patient to know that he has nothing to fear in regard to the operation, and that he will be comfortably propped up in bed with freedom of head movement. Make light of the operation from his point of view, but at the same time impress upon him the necessity for his cooperation in the way you indicate.

He is then sent for a culture. On admission to hospital the night before the operation, I make sure that he has a restful night by giving a capsule of "Hebaral sodium", "Sodium amytal" or "Nembutal". The eye is prepared early the next morning and homatropine (1% solution) is put into the eye about two hours before the operation. I find that the pupil should not be too widely dilated before the operation, and endeavour to obtain about two-thirds dilatation. In my experience, it has been no hindrance to the delivery of the lens, and allows eserine to act more quickly after the operation.

If I find the pupil is too widely dilated when I arrive, I put eserine into the eye at once. You will notice that I differ slightly in this respect from Sinclair, but I am giving you the results of my own experience which has led me to the technique I now put before you. I now commence to use the local anæsthetic, and use a 1% solution of "Pantocain" because it has little or no effect on the size of the pupil.

As regards illumination I use the Lister hand lamp, and endeavour always to have the same specially trained nurse to hold it. I use an ordinary retractor, and do a canthotomy only when the palpebral aperture is too small. I use a periorbital "Novocain" injection in only a few cases, when I feel that the patient will not cooperate with me. I have found that the proper mental preparation of the patient beforehand, and a few words of encouragement during anæsthetization, with that very necessary transfer in subtle ways of your own confidence to the patient, are all that are necessary to secure his very satisfactory cooperation. This method has never yet let me down. Even during the operation I frequently encourage the patient and impress on him the necessity of keeping both eyes open, of looking in a certain position and of not closing the eyes. I always warn patients when about to do the iridectomy. If you feel that you cannot gain sufficient of the patient's confidence to allow you to get such control of his will, then there is only one thing to do, and that is to follow out the methods of paralysing the motor nerve supply to the orbicularis.

Before going on to a description of the actual operation, we shall now deal with the group of unsuitable cases.

The first group of unsuitable cases includes cataracts in juveniles and adults up to the age of about thirty-five. I always hoped it would be a simple operation in these people in order to avoid the slow convalescence and necessity for needlings or curette evacuations, but my experiences and results have been most unsatisfactory, due to the difficulty in breaking the suspensory ligament. The ligament seems to be very much more elastic and firmly attached in people under the age of thirty-five and it becomes less so as age increases after that period. I have attempted six cases up to date with the following results.

A man of twenty-four had an opaque right lens resulting from an explosion and only $^{6}/_{36}$ vision in the other eye, due to a brass intraocular foreign body lying between disk and macula, which was also the result of the explosion.

When I first saw him his vision was $^{6}/_{6}$ partly in the right eye, but he had an early star-shaped traumatic cataract. This rapidly developed and the lens was opaque in six months, but his projection was good. I attempted an intracapsular extraction, expecting it to end early in a capsulectomy, but the capsule hung on tenaciously. I tried hard to sever the suspensory ligament both by direct pull and counterpressure, but it would not break; finally the capsule broke, so I proceeded to a curette evacuation. His projection was poor from the beginning, and finally after several weeks of convalescence I was able to see through and found that he had a complete retinal detachment. I consoled myself with the thought that possibly the traumatic element was a factor in predisposing to this result, but in view of my further experience with this group I think it is always a decided possibility.

My next attempt was in a girl of twenty-one with a bilateral congenital type of cataract. This ended in capsulectomy and curette evacuation, with finally a good result. Here again I experienced the greatest difficulty in attempting to sever the suspensory ligament.

The next case was in a boy of nineteen, with bilateral congenital cataract, which ended in the same way as the last-mentioned case.

My next attempt was in a man of thirty, with an unusual type of progressive cataract which commenced in adolescence. This ended in capsulectomy and curette evacuation with a decidedly slow convalescence and not a very satisfactory result.

The fifth case was in a young man of twenty-five with bilateral congenital cataract. In this case the suspensory ligament appeared to break above, and I felt that we were going to be successful, but then the capsule broke, and in an attempt at curette evacuation vitreous began to appear from above the lens and so it was left. Since then several needlings have been done and the patient is still without good vision.

The sixth case was in a man of thirty-three, with fairly rapidly developing bilateral cataracts, the causation of which was unknown. This case also ended in capsulectomy and curette evacuation, but with a satisfactory result. The suspensory ligament was just as elastic in this case, and seemed able to withstand a lot of pulling without the least sign of breaking.

In patients between the ages of thirty-five and forty I have had several successful intracapsular extractions. One in particular that I can recall was a case of cataract complicating *retinitis pigmentosa*, the only one of this type I have yet attempted. The patient has now $^6/_9$ central vision with a very contracted field. Several cases of this age have ended in capsulectomy and curette evacuation, and I have found that until after the age of fifty some difficulty in breaking the suspensory ligament is to be expected. In this group of cases up to the age of thirty-five I have therefore made up my mind to content myself in future with an intentional capsulectomy and curette evacuation without placing any strain on the suspensory ligament at all. In patients up to the age of forty-five or fifty I should advise you to limit the effort of attempt to break the ligament to a reasonable amount; if it does not break, then finish with capsulectomy *et cetera*.

The next group of unsuitable cases will include exophthalmos and those patients who are inclined to have prominent eyes and in whom, after you have made your corneal incision, there is a definite spontaneous tendency for separation of the edge of the wound with the iris presenting above. Loss of vitreous will almost certainly occur if you persist in intracapsular extraction in this type of case. I have found that patients with a degenerate and fluid vitreous will also give you this valuable warning not to persist.

The most favourable type of case for the performance of the operation is when, after incision, the lens looks, if anything, deeper than before, and the iris lies in position quite naturally.

The third group of unsuitable cases includes the very old and perhaps deaf person whose cooperation for these or some other such reasons can definitely not be relied upon.

I have now reached a stage when I feel I can assess a patient's power of cooperation, and if I have not been able satisfactorily to subordinate his will to mine in this respect, I do not attempt the operation.

THE OPERATION.

With regard to the incision itself, I refer you to Sinclair's description, as I have found it most satisfactory.

I next do the iridectomy and always endeavour to do a peripheral one; this becomes very difficult with a very fully dilated pupil, and is another reason for my efforts to keep the pupil at two-thirds dilatation. I have so far preferred not to do an intracapsular extraction when a complete iridectomy has had to be performed. If for some reason the pupil is unyielding and very rigid when you commence to deliver the lens, the worst that can happen is a capsulectomy and then delivery of the lens in the ordinary way. I find that the more familiar I become with the operation, the fewer such terminations do I experience. One now takes up the intracapsular forceps and decides, after considering everything, what type of extraction one will perform, that is, intracapsular extraction or capsulectomy. In regard to the method of introduction of the forceps, I have nothing to add to the description given by Sinclair himself.

With regard to picking up the lens capsule, I have on occasions had great difficulty in getting the forceps to grasp the capsule. The more mature the lens, the easier it appears to be. I make several attempts, varying the pressure and position of the forceps a little in those that are difficult to pick up. I find that the amount of pressure on the lens necessary to pick up the capsule varies in different cases and can be acquired by experience only. When I have hold of the capsule and have then broken the suspensory ligament, I use an iris repositor for external pressure and conduct the final stages of the operation in the same way as described by Sinclair. After the operation I instill 1% eserine drops, and then apply a double pad and tie. I then insist on leaving the patient quietly at rest on the operating table for twenty minutes before being put back to bed. This, I find, gives ample time for the eserine to contract the pupil sufficiently to obviate the possibility of prolapse.

HÆMATEMESIS DUE TO GASTRIC AND DUODENAL ULCER.

By C. Gordon Shaw,
Melbourne.

The treatment of hæmatemesis due to chronic gastric and duodenal ulcer, like so many questions in gastric surgery, has given rise to views so different and methods of dealing with this emergency so opposed, that one hesitates before he lays down dogmatically what he regards as the correct practice to be pursued. However, I am convinced that the time has come, is in fact overdue, when we should state in unmistakable terms what we regard as the proper treatment for these conditions.

Causes of Hæmatemesis.

Of 86 cases of hæmatemesis occurring in patients admitted to Saint Vincent's Hospital in the last seven years, 32 were due to gastric ulcer, 16 to duodenal ulcer, and 38 to other causes. It is seen that the hæmatemesis in something over half of the cases was due to gastric and duodenal ulcers. These figures correspond roughly with statistics quoted elsewhere. The other causes of hæmatemesis include carcinoma of the stomach, gastritis, cirrhosis of the liver, tumours of the liver, gall-stones, appendicitis, splenic anæmia and septicæmia.

Frequency of Hæmorrhage in Gastric and Duodenal Ulcer.

It is certain that bleeding occurs sooner or later in practically all cases of peptic ulcer, though it may be so slight as to escape notice by the patient; and so a history of this complication is not obtained in the majority of cases.

Of 190 patients with gastric ulcer, 34 or 17·8% had a moderate or severe bleeding, and of 136 patients with duodenal ulcer, 18 or 13·2% had a history of hæmatemesis or of melæna or of both. These figures are probably too low. Pannett states that severe bleeding occurs in 25% of chronic gastric and 20% of chronic duodenal ulcers.

Mortality due to Hæmorrhage.

Opinions differ greatly as to the danger to life associated with hæmorrhage. This is a vitally important point, as the treatment adopted must be based on whether bleeding from a chronic peptic ulcer may be fatal or not. Paterson and Hurst both state that they have never seen a death from hæmorrhage, and Maclean states that with proper

treatment hæmorrhage is rarely fatal. Walton states that in gastric ulcer the hæmorrhage is either so severe that death occurs before operation is possible, or else it ceases spontaneously, but that in duodenal ulcer hæmorrhage is more serious.

If these facts are correct, it follows that operative treatment is wrong, and that these patients should be treated medically.

On the other hand, Sherren considers that death from this cause is common, and quotes the London Hospital mortality of patients admitted with hæmatemesis as being 12·25% among men, and 6·5% among women; also of 55 deaths from chronic ulcer, 25 were due to hæmorrhage.

Among 330 patients with peptic ulcer admitted to Saint Vincent's Hospital, Melbourne, 37 had hæmorrhage, which was either moderate or severe during their stay in hospital; and of these, 35 were treated on conservative lines, and of the 35 five died from bleeding. Two patients were operated on. In one instance a gastric exclusion and in the other a gastro-enterostomy was performed, and both patients recovered. The condition of many of those who were treated medically was very critical, and because of repeated hæmorrhages their fate hung in the balance for several days.

I think that it must be accepted that hæmorrhage is a dangerous complication of chronic ulcer, and that the opinion that death does not occur with medical treatment is unwarranted.

It is not my purpose to consider hæmorrhage from acute ulcers and erosions, but I may say that the bleeding in these cases comes from a small submucous vessel, that it is often very copious, reducing the patient in a short time to a critical condition, but that it does not tend to recur, and this I regard as an important feature. The treatment is undoubtedly medical, and the mortality is very low, being between 1% and 2%. The mortality following operation is probably about 60%.

The considerations which should guide us in the treatment of chronic peptic ulcer are, however, very different. The artery from which the bleeding occurs is a large one, the right and left gastric, the gastro-duodenal, the pancreatico-duodenal, the right and left gastro-epiploic and the splenic arteries being the arteries which may be eroded. Further, the bleeding is generally recurrent, and in this respect it differs in no way from secondary hæmorrhage elsewhere.

The clinical features of the case are no less important. I have never seen a patient die as a result of the first bleeding, though it is stated that in 54 cases rapid death occurred in eight. In some of these cases at least it was the splenic artery that was involved. The patient generally recovers from the first hæmorrhage, but after two or three days further bleeding takes place; this recurs several times, so that an operation cannot be undertaken with any reasonable hope of success, and death may occur within a few days of the initial bleeding.

It must also be borne in mind that sometimes the hæmorrhage is small at first, but that the recurrent hæmorrhages are larger and endanger life.

In a certain number of cases the bleeding is moderate, and presumably for this reason it does not cease, but goes on day after day till death ensues.

TREATMENT.

Consideration of these facts indicate very definitely to my mind the correct line of treatment.

In a small number of cases death occurs very rapidly as a result of a copious hæmorrhage, and in these no treatment will be of any avail. In another small group the hæmorrhage is moderate in degree, but goes on day after day until the patient is reduced to a critical condition. It is clear that operation must not be delayed till it becomes a dangerous undertaking; it should be performed during the first twenty-four to forty-eight hours, and under this condition will be a life-saving procedure.

In the majority of cases a severe hæmorrhage occurs, and the patient's condition is serious; the bleeding temporarily ceases, and the condition of the patient improves. I submit that operation should be performed just as soon as the condition of the patient improves sufficiently to allow its being undertaken with reasonable hope of success, after the first hæmorrhage and before the second hæmorrhage takes place, and that is as a rule from twenty-four to sixty hours after the first bleeding.

It is quite obviously wrong to wait till repeated hæmorrhages have rendered any surgical procedure a forlorn hope, and it is this practice which has been responsible for the belief that surgery is unsuited to these cases.

Nature of Operation.—In all severe cases I consider that the operation should be regarded as a life-saving one, and that the only interference should be that which is necessary to stop bleeding. In the case of a chronic gastric ulcer, provided the ulcer is not densely adherent to the pancreas or liver, this is easily and rapidly done by under-running and ligaturing all the vessels which enter the ulcer. If the ulcer is in its most common site on the lesser curvature, the right and left gastric arteries are ligatured, and also any other large vessel which may be entering the curvature.

If the ulcer is adherent, it may be necessary to cut through the stomach wall at the periphery of the ulcer, freeing the stomach, and leaving the base of the ulcer attached; the opening in the stomach is then closed in the usual way, and the abdomen is closed with drainage to the site of the ulcer. It has been advised to cauterize the ulcer and then to suture the stomach after the method introduced by Balfour in the treatment of chronic gastric ulcer. It seems to me that this procedure unnecessarily prolongs the operation, and also that there is a risk of post-operative secondary hæmorrhage.

Finsterer carries out an extensive resection of the stomach; in fact, he treats the condition exactly as he would treat it if it were not complicated by hæmorrhage. He always operates with local anæsthesia.

I regard the performance of a gastro-enterostomy only, as inadequate, if it is done with the object of stopping hæmorrhage.

In the case of a chronic duodenal ulcer, provided the ulcer is on the anterior surface, it should be turned in by three rows of deeply placed Lembert sutures, so as to obstruct the duodenum, as well as to compress the vessels entering the ulcer; a gastro-enterostomy should then be performed.

Where the ulcer is on the deep surface, the bleeding will be more difficult to deal with, and it may be necessary to open the duodenum and to under-run the bleeding vessel with a ligature, as in a case successfully operated on by Dunhill. At the end of the operation, and not at the beginning, a blood transfusion should be given.

RESULTS OF TREATMENT.

For the last three years all patients admitted under my care at Caulfield Repatriation Hospital and all private patients have been treated on these principles. They include three patients with severe hæmorrhage from an acute ulcer which were treated conservatively, all of whom recovered. There were also seven cases of severe hæmorrhage from chronic gastric and duodenal ulcer which were treated by operation. In none of these did any further bleeding occur after the operation, and all of the patients, though they had lost a very large quantity of blood, had an uneventful recovery. One of these cases was of special interest.

It was that of a man, aged forty-seven years, who was almost exsanguinated by a hæmorrhage from a chronic ulcer on the lesser curvature. At operation, performed about forty-eight hours after the hæmorrhage, a very large ulcer was found, and the right and left gastric arteries and several other large vessels were ligatured. Six weeks later an operation was performed with the object of doing a partial gastrectomy. It was found that the ulcer was about one-quarter of its former size, and the operation was performed very much more easily than it could have been performed at the first operation.

CONCLUSIONS.

1. All patients with hæmorrhage from a gastric or duodenal ulcer admitted to a public hospital should be put in a surgical ward, and should be under the care of a surgeon and not of a physician.

2. Hæmorrhage from a chronic gastric or duodenal ulcer should be treated by operation just as soon as the condition of the patient permits.

3. The operation should have as its object the stopping of hæmorrhage only.

4. A blood transfusion should be given at the end of the operation.

SOME OBSERVATIONS ON THE PATHOLOGY OF APPENDICITIS.

By Alfred J. Trinca,
Melbourne.

The numerous theories that have been advanced to explain the ætiology and pathology of appendicitis are in themselves evidence of the uncertainty which exists in regard to this common surgical disease. Age, sex, heredity, familial tendency, trauma, foreign bodies, intestinal parasites, diet, neuro-angiospasm, hæmatogenous and enterogenous infection have all been put forward as factors.

Authors of modern textbooks on pathology and surgery accept the view that acute appendicitis is a primary inflammation commencing in the mucosa, very often in a small lesion, and that the essential cause is a bacterial infection, most commonly enterogenous in origin, but possibly hæmatogenous. Mild degrees of inflammation are described as catarrh, more severe as gangrenous inflammation.[1]

Wilkie[2] drew attention to the importance of obstruction as a cause of a certain type of appendicitis, and following on this has arisen the view that gangrene can occur only in the presence of obstruction, or that obstruction is the basis of appendicitis in the majority of cases.[3]

It is my purpose in this paper to criticize the accepted pathology of appendicitis, and to formulate a theory of the mechanism by which pathological changes are produced in the appendix, and to describe what is considered as the essential pathological basis for the changes occurring in this organ in so-called inflammation.

Bacterial Infection.

If acute appendicitis be the result of bacterial invasion, it is remarkable that no specific organism has been discovered. To quote McCallum:[4]

> It is a strange thing that in spite of the bacteriological investigations that have been carried out in the problem of appendicitis, no specific prevailing organism has been found. The *Bacillus coli*, streptococci, anaerobes are all singly or in conjunction found, in fact the intestinal flora . . . .

In the various classifications of appendicitis we meet the following terms: acute catarrhal appendicitis, acute gangrenous appendicitis, acute perforated appendicitis, acute and chronic obstructive appendicitis, chronic appendicitis.

Boyd[5] shows how unsatisfactory the position is when he states:

> It is very difficult to hit on a classification of appendicitis which will be satisfying both to the pathologist and clinician.

ACUTE CATARRHAL APPENDICITIS.

The appendix removed early in the course of an attack of acute "apendicitis" shows a swollen reddened mucosa with or without exudate, and congestion of the subperitoneal vessels. It is described as acute catarrh.

The term "catarrh" in pathology means an inflammation of a mucous membrane, and is characterized by a tendency to spread in its acute stages. A nasal catarrh quickly becomes a naso-pharyngeal and often a naso-pharyngo-tracheo-bronchial catarrh. Acute gastric catarrh usually becomes gastro-intestinal and so on. Why then does acute catarrh of the appendix remain confined to the appendix without involving the caecum, which is developmentally the same and almost structurally identical? And why does one so often see catarrh confined to only a portion of the appendix?

ACUTE GANGRENOUS APPENDICITIS.

In acute gangrenous appendicitis a portion or the whole of the appendix rapidly becomes gangrenous. Sometimes only the mucosa is found involved, more often all the coats. Is this condition the result of infection by a gangrene-producing organism, or is it the response to an infection so intense that gangrene results? If due to either of these causes, why does the process stop short at the appendix and not spread to the caecum. If it involves the caecum, why is the gangrene confined to a small area around the base of the appendix, or why is it so frequently confined to a portion only of the appendix? In the latter event, what is the explanation of the sharp line of demarcation between the healthy and gangrenous portions? (See Figure I.) Does the inflammation suddenly stop short at this line and the healthy appendix beyond it suddenly develop a resistance? Surely some other explanation besides that of a primary inflammation must be given in both catarrhal and gangrenous appendicitis.

Gangrene in pathology, whether infective or not, has a single fundamental cause, and

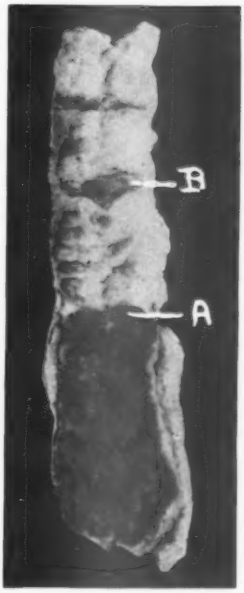

FIGURE I. Appendix, natural size, removed from a boy, aged fifteen, ten hours after onset of symptoms. The terminal portion shows gangrene of the mucosa sharply marked off from the unaffected portion at *A*. *B* shows an area of venous congestion due to obstruction of the blood supply by ligation of the meso-appendix.

that is interference with the blood supply. The failure to find a gangrene-producing organism in the majority of cases of gangrenous appendicitis forces us to look for a non-infective cause.

There appears to be only one answer to these questions: catarrh and gangrene are not due to a primary inflammation.

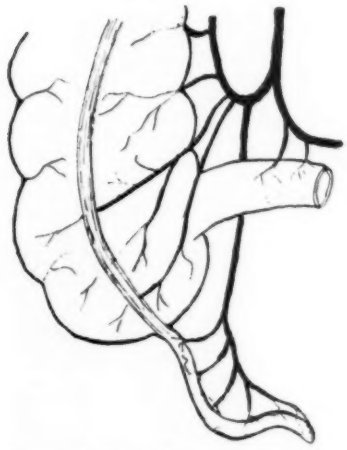

FIGURE II. Appendiceal artery supplying appendix only.

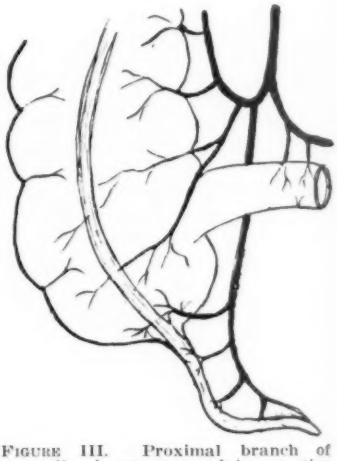

FIGURE III. Proximal branch of appendiceal artery supplying portion of caecum.

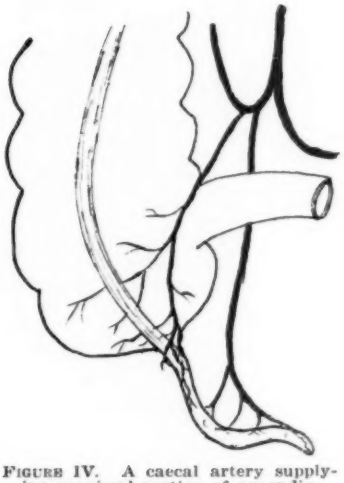

FIGURE IV. A caecal artery supplying proximal portion of appendix.

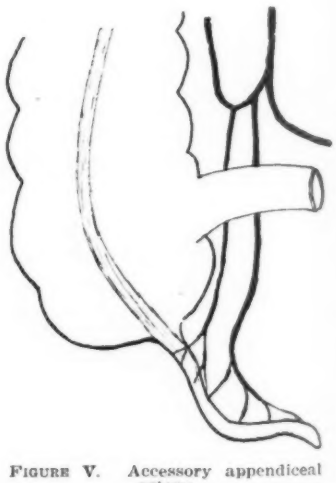

FIGURE V. Accessory appendiceal artery.

ACUTE PERFORATING APPENDICITIS.

Acute perforating appendicitis is due to two causes: either a rapidly softening gangrenous appendix bursts in one or more places, particularly so when obstructed, or the presence of a fæcolith produces pressure necrosis with perforation.

THE BLOOD SUPPLY OF THE APPENDIX.

There are many variations in the blood supply of the appendix.[6][7] (See Figures II, III, IV, V and VI.) The main supply in most cases is from the appendiceal artery which usually arises from the posterior ileo-caecal or less commonly from the ileo-colic artery direct or from one of the terminal iliac branches of the superior mesenteric. It passes behind the terminal part of the ileum, and then runs between the layers of the meso-appendix.

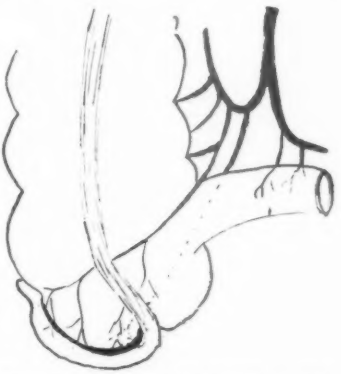

FIGURE VI. Appendix bound to wall of caecum and supplied by small caecal arteries. Appendiceal artery rudimentary.

It may or may not run to the tip of the appendix. It usually gives off from three to five branches, which anastomose in a variable manner as regards completeness in the wall of the appendix. Anastomotic loops in the meso-appendix itself are not common. Injection of the main branches of the appendiceal artery will often reveal in the mucosa a lowly vascularized or anæmic area between the areas of distribution of these vessels. In other words, at certain points these branches are end arteries, and there are zones between the areas supplied by these individual branches where the wall of the appendix, particularly the mucosa, is poorly supplied with blood. On opening a normal appendix which has been removed at operation, one can see areas of venous congestion or hæmorrhage resulting from ligation of the vessels in the meso-appendix. These areas are strictly localized and not confluent, and this appearance adds confirmation to the statement that poor anastomosis exists in the mucosa between the branches of the appendiceal artery. In cases in which the meso-appendix ends some distance from the tip, the terminal portion is often relatively insufficiently supplied with blood. The proximal portion of the appendix and the adjacent caecum have a variable blood supply. The portion of caecum surrounding the base of the appendix may be dependent for its blood supply on the proximal branch of the appendiceal artery, and the proximal portion of the appendix may derive its blood supply from one of the caecal arteries. In a certain percentage of cases there is an accessory appendiceal artery from the posterior caecal which supplies

this proximal portion. The appendiceal artery itself is a single end artery; in other words, it does not anastomose with any other branches of the ileo-colic. Having reached the appendix, each branch of the appendiceal artery subdivides into two or more sub-branches before penetrating the coats of the appendix. Then, having pierced the serosa, it forms two main systems, one of which encircles the appendix in a tortuous fashion under the serosa, and the other—the larger—which forms the basis of blood supply for the other coats. The branches from this system are very fine and variable. It would appear that the peritoneal and subperitoneal regions compared with the rest of the wall are disproportionately supplied with blood. This is seen in certain cases of partial gangrene of the appendix where the appearance of the peritoneal coat belies the amount of destructive pathological change seen in the mucosa and muscle coats when the appendix is slit open.

The meso-appendix is richly supplied with blood, and the number of vessels supplying the fat seem to be out of proportion to that necessary to nourish a similar amount of fat. When the appendix is retro-peritoneal or bound to the lateral wall of the caecum without any obvious mesentery, the appendiceal artery tends to be rudimentary, or it may be absent and the appendix dependent for its blood supply on the small arteries that supply the caecum.

THE POSITION OF THE APPENDIX.

The cadaveric position of the appendix is not the same as that in life.[8] It can be shown that the position of the free appendix is dependent on the position and state of distension of the caecum at any particular time. Experiments conducted on the cadaver show that distension of the caecum can alter the position of the appendix from a pelvic or splenic position to a retro-caecal or para-caecal position. In fact, any mobile appendix with a well developed mesentery can adopt almost any position.

Distension of the caecum in the living subject produced by forcing air out of the transverse colon into the caecum will produce the same effects, and the appendix can be seen to rotate on its axis or bend over on its mesentery, or develop a kink. The latter particularly happens if portion of the appendix is bound down by a developmental or adventitious adhesion (Figures VII and VIII).

THEORY OF CAUSATION OF "ACUTE APPENDICITIS".

It is our contention that the fundamental basis of the pathology of appendicitis is interference with the blood supply. It will be admitted that an exciting cause of interference with the blood supply of the appendix need not always be of the same intensity or of the same duration, and that a temporary interference of the blood supply will not produce death of tissue, but that a prolonged interference will.

Any organ or tumour with a pedicle is liable to undergo torsion and any structure of which the connexions are the equivalent of a pedicle, such as the sigmoid colon with a contracted mesenteric base, is

FIGURE VII. Position of appendix before artificial distension. Note loose meso-appendix.

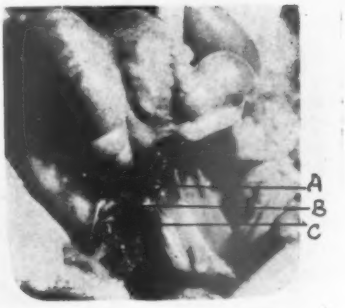

FIGURE VIII. State and position of appendix after distension of caecum in same subject as Figure VII. Note acute kink at *A*, dilatation at *B*. Appendix rotated on itself at *C* and tension along border of meso-appendix.

liable to undergo torsion in a similar manner. Occasionally the tube and ovary undergo torsion with interference with the blood supply and actual gangrene. A mobile undescended testis may do the same thing; a pedunculated tumour has a great tendency to twist. What is the physical process which causes torsion? Is it intestinal distension, peristaltic movement, or the movement of other organs, such as the filling or emptying of a bladder? Torsion of the structures mentioned is an indisputable fact. Why then cannot the same event take place in an appendix with a long mesentery, or a mesentery with a narrow base?

Proof of a certain degree of twisting can be observed occasionally by the surgeon whilst removing a pathological appendix. The appendix can often be felt to rotate or unwind before it presents itself through the wound, and this can be particularly demonstrated if the appendix be delivered in an indirect manner by pulling on the caecum (Figure IX).

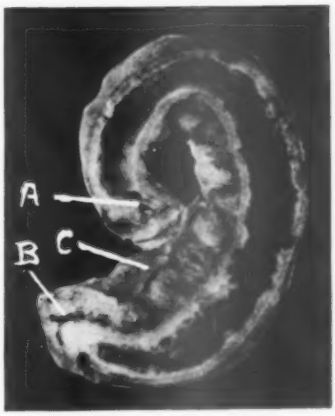

FIGURE IX. Appendix, natural size, showing the effects of torsion, removed from a girl, aged eight years, sixteen hours after the onset of symptoms. Complete gangrene has occurred. *A* is the tip and *B* the base, showing recent constriction due to torsion. The appendix was twisted two complete turns at this point. *C* is the mesentery.

Torsion is an example of the process which could produce the most complete interference with blood

supply of the appendix, but it is not contended that this occurs in any but the minority of cases of vascular interference. The blood supply can be interfered with in other ways. The appendix can be bent on itself and kept fixed in this position by the pressure of hollow viscera. The appendiceal artery goes behind the terminal part of the ileum and one can imagine in an appendix that is held down by developmental or other adhesions, an interference with its blood supply by pressure on its artery by a distended terminal portion of the ileum. An appendix forced into a retro-caecal fossa and then held there by abnormal caecal pressure could similarly suffer from ischæmia. The retro-peritoneal appendix or the appendix bound to the wall of the caecum can suffer interference with its blood supply by pressure between the caecum and the parietes.

It is our view that as the result of distension of the caecum associated with normal or irregular peristaltic movements, the appendix is either twisted on its mesentery, or kinked, or, if in a fixed position, compressed by a distended caecum against the unyielding parietes with a production of temporary or prolonged ischæmia.

It can be understood that only certain individuals can be the subjects of appendicitis, and that the incidence of appendicitis and the severity or nature of any particular attack will depend on anatomical factors, for example, the development and nature of the meso-appendix, peritoneal relationships or fixation of the appendix, congenital anomalies, and the particular type of the blood supply of the appendix. In other words, distension will produce its pathological effects on the appendix only when the right set of anatomical conditions are present.

THE PATHOLOGICAL EFFECT OF INTERFERENCE WITH THE BLOOD SUPPLY OF THE APPENDIX.

Partial or temporary interference with the blood supply produces ischæmia followed by congestion or tissue stagnation in varying degree, and produces the phenomena of so-called catarrh. In a certain number of cases the interference with nutrition will be serious enough to allow of lowering of resistance with a secondary invasion by any intestinal flora present. This may be confined to the mucosa or extend through all the coats, and produce either an indirect or direct involvement of the peritoneal cavity. A longer period of anæmia and vascular stasis will produce actual death of some or all of the coats, and the condition described as gangrenous appendicitis, which will be further aggravated by bacterial invasion. According to whether the main appendiceal artery or one or more of its branches is involved, so will the gangrene be complete or confined to a portion of the appendix. If the interference is of short duration, complete recovery can occur; if of longer duration, some damage is inevitable. Desquamated epithelium may not be restored, the secondary inflammation may result in fibrosis, stenosis, atrophy, atonicity *et cetera*, and the formation of adhesions, and

this can pave the way for a subsequent attack of obstructive appendicitis, or make a future attack more severe in its results.

In perforation by a fæcolith there is interference with the blood supply or nutrition of a local patch of the wall of the appendix and ulceration or pressure gangrene occurs, the secondary inflammation following the gangrene increases the pressure, and so the area of gangrene tends to be larger than the area of the fæcolith.

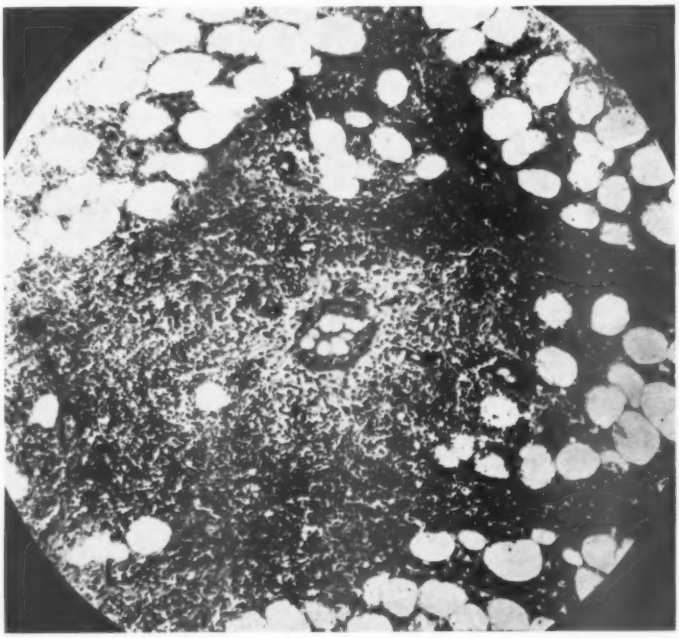

FIGURE X. Photomicrograph of section of meso-appendix in Figure IX just beyond point of twisting, showing hæmorrhage and leucocyte infiltration around a blood vessel resembling the appearances found in hæmorrhagic infarct. No microorganisms could be demonstrated in this area.

An interesting observation which bears out the argument that interference with the blood supply is the cause of gangrene, is that the appendix deriving its blood supply from the wall of the caecum is usually gangrenous throughout its length. In the case of the retro-caecal appendix, if it is not entirely gangrenous, only the base escapes, and this survives when it has a separate blood supply from one of the caecal arteries.

In regard to the effect on the mesentery, microscopic section shows œdema and a condition resembling hæmorrhagic infarct (Figure X). No

organisms can be demonstrated in the area of hæmorrhage, and leucocytic infiltration is only seen at a later stage. One would expect an early invasion of the mesentery by organisms if the condition were the result of a primary inflammation in the appendix.

ACUTE OBSTRUCTIVE APPENDICITIS.

Acute obstructive appendicitis is either due to localized trauma having caused stenosis, atonicity, and an unhealthy mucosa, the result of a previous attack, or for developmental reasons either in or outside the appendix a stricture has developed. Fæcoliths often develop in such appendices, and their presence and the accumulation of mucus increase the tension in the lumen of the appendix, and any interference with blood supply or attack of inflammation will have a much more intensive effect.

CHRONIC APPENDICITIS.

It can be understood how one or more attacks of congestion or partial gangrene can result in fibrosis, atrophy of mucosa, stenosis, obliteration, stricture *et cetera*, or in a milder degree produce atonicity with stasis and ultimate fæcolith production. Our conception of so-called chronic appendicitis is that, apart from developmental anomalies, most of the conditions found are the result of a previous attack of vascular disturbance, and not a condition resulting from a chronic primary infection starting in the mucosa.

ÆTIOLOGY.

It would appear that appendicitis is a disease of modern civilization, and was not known before the latter half of the nineteenth century. During this period of history large quantities of meat and refined cereal foods were added to the diet of civilized people, with a corresponding elimination of cellulose. That diet is an important factor is shown by the fact that in many uncivilized tribes appendicitis is unknown until these natives adopt the European diet, when it becomes comparatively common.

Lucas-Championnière,[9] in an analysis of twenty-two thousand patients among Roumanian peasants, who lived mostly on vegetables, found only one case of appendicitis. The Roumanians living in the cities chiefly on animal diet, were frequently affected, one case of appendicitis occurring among every two hundred and twenty-one patients. Appendicitis is rare among the Japanese and Indians, and unknown among the Arabs, who live in tribes and are vegetable eaters, but it is very prevalent among the Arabs in the cities where meat is the chief diet.

It is possible that modern civilized diet, with its associated overeating and irregularity in meals and life generally, plays its part in the ætiology of appendicitis, and that intestinal indigestion, with overloading and distension of the caecum, and perhaps abnormal peristalsis are the exciting causes of the interference with the blood supply which

produces so-called appendicitis where anatomical conditions are favourable.

It is a well known fact that the administration of a purgative in the early stages of an attack of appendicitis will tend to produce gangrene and perforation. Purgatives increase peristalsis and produce distension, and the aggravation produced by them to some extent supports the theory of a mechanical factor in the causation of appendicitis.

SUMMARY.

1. The accepted pathology is criticized.

2. Appendicitis is not a primary inflammation.

3. Anatomical and pathological studies suggest that apart from developmental anomalies the true cause of acute appendicitis is interference with the blood supply.

4. Chronic and other forms of appendicitis are the sequelæ of one or more acute attacks.

5. Modern civilization and diet are the predisposing factors.

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EXPERIMENTAL UNILATERAL URETERO-INTESTINAL ANASTOMOSIS.

THREE YEARS' SURVIVAL AFTER UNILATERAL URETERO-COLOSTOMY FOLLOWED BY OPPOSITE NEPHRECTOMY.

By ADOLPH BOLLIGER AND P. N. WALKER-TAYLOR.

[From the Gordon Craig Urological Research Laboratory, Department of Surgery, University of Sydney.]

IN a recent paper Kirwin[1] points out that after three years of experimental work he has been unable to obtain in the dog a satisfactory ureteral implant with Coffey's method, as well as with a technique of his own. In all of his experiments the animal revealed serious disorganization of the urinary apparatus if sacrificed from two to four months after the unilateral or bilateral implantation. In view of these findings, which are also supported by those of Sisk, Weir and O'Brien,[2] it may be permissible to refer again to results obtained in this laboratory with experimental transplantations of ureters into the intestine,[3][4] and particularly to give the final result of an experiment which was not completed when the previous experiments were reported.

A brown male sheepdog, with a body weight of twelve kilograms, was operated on on March 8, 1930. The right ureter was implanted into the lower part of the colon, the so-called "open tunnel" method being used.[3] Sixteen days later left nephrectomy was performed. The dog quickly recovered from the operations, gained weight, and soon became accustomed to the new outlet of urine through the bowels, which it emptied on an average about every three hours. Although in perfect health for the next two years, the animal constantly showed an elevated blood urea, fluctuating mostly between 50 and 70 milligrammes per 100 cubic centimetres of blood, but values as high as 116 milligrammes were observed. The last observation, made on March 12, 1932, was 99 milligrammes.

Owing to the temporary absence of both the authors from Sydney, the animal was handed over to the School of Veterinary Science, and Dr. R. M. C. Gunn kept the dog under observation. However, no further determinations of the blood urea content were made. Dr. Gunn reported that the dog remained in perfect health up to the beginning of March, 1933; it then began to lose weight, refused its food, and died shortly afterwards on March 24, 1933.

At autopsy, the right kidney, together with its implanted ureter and the lower intestinal tract, was removed *in toto*. The specimen so obtained is depicted in Figure I, together with the left kidney which had been removed three years previously, shortly after the uretero-colostomy. The kidney the ureter of which had been implanted was about two and a half times the size of the kidney which had been removed by nephrectomy. On sectioning this large kidney, abscesses were seen in the cortex and medulla, as well as large hæmorrhagic areas which occupied the greater part of the medulla. The pelvis of this kidney

was moderately dilated, and was found to be filled with thick pus. However, its lumen was found to be patent throughout.

The outstanding feature of this case is the survival for three years of an animal with only one kidney the ureter of which had been implanted into the colon. During the first two years of the experiment the blood urea content was raised continuously, as shown by numerous blood urea determinations. This undoubtedly also must have been the case during the third year of the experiment, although no actual determinations were performed.

FIGURE 1. Unilateral uretero-colostomy, followed by opposite nephrectomy, of three years' standing. The small kidney was removed sixteen days after uretero-colostomy.

As pointed out in a previous paper, the raised blood urea was due to reabsorption of urinary constituents in the lower part of the bowel. This anomalous condition also led to hypertrophy of the remaining kidney. A somewhat similar result was recently obtained by Bollman and Mann[5] after unilateral uretero-jejunostomy and uretero-duodenostomy, followed by removal of the other kidney. These authors found that the hypertrophy of the remaining kidney, after nephrectomy, was considerably greater when the ureter was implanted into the bowel than that observed following simple nephrectomy without subsequent transplantation. They attributed this to the accumulation of urinary products in the blood due to the absorption of urine from the upper part of the intestinal tract. In these short-period experiments the kidney the ureter of which had been implanted increased in weight from 60% to 100% within nine days, as compared with the kidney removed by nephrectomy. In the long-period experiment described in this

paper the increase of the weight of the kidney as compared to that of its fellow removed three years previously, amounts approximately to 150%, the weight of the kidney removed by nephrectomy being 130 grammes. However, we are well aware of the fact that the infection of the hypertrophied kidney somewhat obscures the picture, but an examination of the gross specimen well demonstrates the unusual increase in size.

It has been pointed out by Kirwin[1] that the dog is by no means the animal best suited for this type of experimental work; the horizontal position of its body with the subsequent stagnation of urine in the whole of the lower part of the intestinal tract would seem to favour ascending infection to a greater degree than in human beings. However, the experiment reported in this paper, together with those mentioned on previous occasions, demonstrates the fact that ureteral implantations in the dog may be approximately as successful as in human beings if one considers the much shorter span of life of a dog compared with that of man. The final outcome is probably the same in both species, provided no other disease interferes. Obvious infection at some time or other will occur in the kidney the ureter of which has been implanted, and the progress of this infection will ultimately lead to death, if the animal depends solely on the proper functioning of this kidney.

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SOME USES AND ABUSES OF LUMBAR AND VENTRICULAR PUNCTURE.

By I. Douglas Miller,
Sydney.

Lumbar and ventricular punctures are procedures of such great value in certain conditions, and their misuse is apt to be followed by such grave misadventure or even disaster, that as far as possible an effort should be made to define their uses and abuses with some accuracy.

Lumbar Puncture.

In dealing with possible surgical conditions of the brain and cord, it should be a routine to use a manometer whenever lumbar puncture is done. A small lumbar puncture needle of the Harris type is the best. Large needles are apt to cause bleeding and a certain amount of pain both during and after the puncture. The simplest form of manometer is a length of fine rubber tubing connected by an adaptor to the end of the lumbar puncture needle, and by the other end to a small glass U tube which is allowed to hang over the edge of a test tube. After the puncture is made the fluid runs into the tube and after its level has been stabilized, the height of the column is measured off on a rule. (See accompanying figure.)

Lumbar Puncture in Head Injuries.

Lumbar puncture in head injuries has indeed but a limited application, and it is in this field above all others that its use is often not guided by a consideration of the underlying pathological condition. In the severe head injury, when we find a patient who is deeply comatose, cyanosed and breathing stertorously, there is no procedure which will influence the almost invariably fatal course. In these cases lumbar puncture is absolutely useless.

The next type is the patient who is admitted to hospital showing signs of recovery, and who usually progresses steadily to complete recovery of consciousness. In this type of case there is no indication for lumbar puncture, for here during the first twenty-four hours there is a state of affairs which can in no way be benefited by a sudden dehydration. The same applies to the use of hypertonic saline solution during this period of recovery.

In the third type of patient, namely, the one who is admitted in a semicomatose condition, and who shows no tendency either to recovery or retrogression, we should not be tempted to assume that this condition

is probably due primarily to increased intracranial pressure and therefore likely to be relieved by lumbar puncture, or the administration of hypertonic saline solution. So far from being beneficial, these procedures are often dangerous, owing to a sudden disturbance of intracranial balance, and at the best are usually disappointing. It may be stated that if one is tempted to use such dehydrating methods, it should be done only with cerebro-spinal fluid manometric readings as the indication and the guide to the amount of fluid to be withdrawn. This

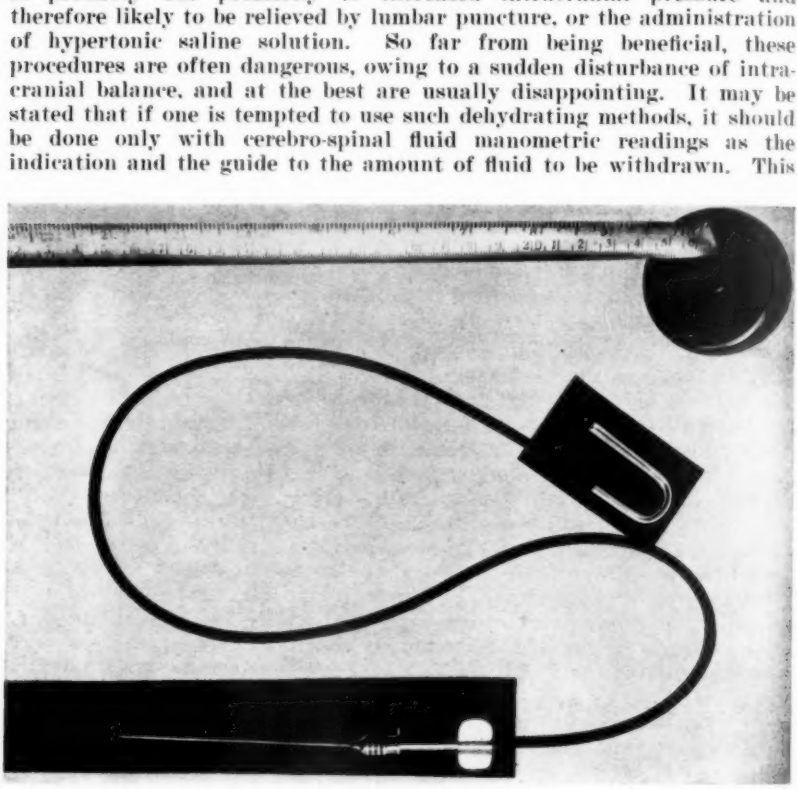

Figure showing manometer for use in lumbar puncture.

condition is then very difficult to fulfil, inasmuch as manometric readings made upon a restless patient are quite unreliable, and there are but few patients in this class who would not be restless under the stimulus of a lumbar puncture.

It is obvious that uncontrolled withdrawal of fluid may be most dangerous, and in the case of massive extradural hæmorrhage may precipitate a sudden disaster.

Taking these points into consideration, and trying to deduce from them some safe working rule which may be generally followed, it may be said that lumbar puncture is contraindicated during the first twenty-four hours following a head injury.

After the first twenty-four hours, that is in the stage of reactionary œdema, lumbar puncture is safer and may have some use. It will often

prove an effective measure in the cerebral œdema which is slow to recover. It should always be controlled by a manometer, and fluid should be withdrawn only until the pressure approximates to normal. It is as well to remember that low pressures may be present after head injuries, and withdrawal of fluid in these cases will not be beneficial. Likewise, if in the stage of recovery dehydration by saline solution is to be used, it is advisable to ascertain the state of intracranial pressure by lumbar puncture before the saline treatment is commenced. Sometimes lumbar puncture is very beneficial, but on the whole it is very disappointing. If lumbar puncture brings about improvement it may be frequently repeated.

Lumbar Puncture in Intracranial Tumour.

In the case of suspected intracranial tumour, lumbar puncture is contraindicated if there are signs of raised intracranial tension. It may easily prove disastrous, and commonly intensifies the headache which may become very severe in the occipital region. This will apply only to cases in which fluid is injudiciously withdrawn, so that if a manometer is used as an indicator no damage is likely to be done.

In dealing with tumours in the posterior fossa, lumbar puncture is very apt to precipitate an acute tonsillar herniation through the *foramen magnum*. When it is permissible, however, considerable information may be obtained from pressure readings and chemical and serological reactions of the fluid.

In suspected abscess of the brain one should be more than usually cautious in withdrawing fluid.

As to lumbar puncture followed by air replacement, this should never be done in the presence of high intracranial tension.

Lumbar Puncture in Spinal Tumour.

In addition to the recognized signs of spinal block obtainable by lumbar puncture, great information as to localization may be obtained from successive lumbar punctures made with a fine needle and the greatest of care in different spaces in an ascending order. The level at which yellow fluid first appears may then be found.

Lumbar Puncture after Intracranial Operations.

It is as well to remember that lumbar puncture may be a most useful post-operative method of reducing tension on an osteoplastic flap. Particularly is it useful in reducing pressure on the flap after posterior fossa operations.

VENTRICULAR PUNCTURE.

Ventricular puncture is not subject to quite the same dangers as lumbar puncture, particularly as it is unlikely to be undertaken so perfunctorily.

In suspected or in unlocalized tumour the procedure is of great value, apart altogether from the replacement of air. Bilateral ventricular

tap may determine a symmetrical ventricular enlargement and in this way decide the presence of a median tumour.

It is worth remembering that even when the fluid is not actively replaced by air, some air will enter by the needle as soon as the fluid ceases to flow. This may be quite a small amount, but nevertheless one may get sufficient shadow to show a gross ventricular shift. In the presence of some increase in intracranial tension this procedure may give much information and is certainly less dangerous than ventriculography.

For deliberate ventriculography it is usual to make two burr holes, though one might be quite effective for removal of the fluid and replacement of oxygen or air. It is as well to withdraw almost all the fluid, as imperfectly filled ventricles may give anomalous appearances. The most important picture is that taken with the patient in the brow-up position, and the most important finding is a lateral shift.

This method of ventriculography should be done as a separate procedure only when there is no increase in intracranial tension. Even then it is a good routine measure to puncture the ventricle a few hours later and to allow the air to bubble out. In the presence of a high increase in tension, as evidenced by papilloedema and severe headache, ventriculography should be done only as the first step of the subsequent operation. After the pictures have been made and examined, the patient should proceed back to the theatre and the operation be undertaken. Failure to carry out this sometimes arduous routine will sooner or later end in disaster.

A very important use for ventricular puncture is found when performing any posterior fossa operation. A preliminary puncture of the ventricle made under the retracted cross bow incision, will greatly reduce the posterior fossa tension and the amount of venous bleeding encountered during the approach. A useful device is a short ventricular needle with a flange at the base. This needle can be inserted into the ventricle and left in place. The flange rests on the skull, and the fluid can trickle away during operation without danger of the needle being knocked.

In supratentorial operations also a ventricular puncture, made even prior to reflecting the dural flap, will often obviate the danger of a sudden bursting out of the brain. This manœuvre is apt to be particularly helpful in some decompression operations when the sudden cerebral bulge becomes an embarrassment. It is a much safer procedure than the intravenous administration of hypertonic saline solution, which is apt to be followed by a reactionary swelling of a severe grade, and which has for this reason been generally discontinued. It is worth remembering that although the routine ventricular tap is made into the occipital horns, the ventricles may conveniently be tapped elsewhere, and therefore from any bone flap exposure. In one case we were unable to strike either occipital horn, owing, as we found later, to the presence of a bilateral tumour, but on making a burr hole over the frontal horn the ventricle was readily entered and good pictures were obtained.

OSTEOCHONDRITIS.

By JOHN HOETS,
Sydney.

UNDER the generic term osteochondritis have come to be grouped all those conditions affecting the ossification centres and epiphyseal plates of growing bone which were called after the men who first described them. Osgood, Legge, Schlatter, Köhler, Calvé, Perthes, Freiberg, Delahaye and Bridgeman were amongst the earlier writers on the subject.

The condition has been noted in more than thirty different situations, the best known being *osteochondritis deformans juvenilis coxæ* of Calvé, Legge and Perthes, epiphysitis of the tibial tubercle (Osgood and Schlatter), Köhler's disease of the tarsal scaphoid, and that condition to which I wish particularly to refer, namely, *osteochondritis deformans juvenilis dorsi.*

In 1921 Schuerman described a vertebral epiphysitis, and in 1925 Calvé noted two cases of a peculiar affection of the vertebral bodies. X rays have been responsible for the discovery of these conditions, and in the case of the hip joint the recognition of what is generally known as Perthes's disease, as a separate entity and distinct from tuberculosis, made possible a more definite differential diagnosis and a more confident prognosis in the former than had been previously possible.

It also made clear the reason why some patients whose condition was diagnosed as tuberculosis of the hip recovered with good range of movement, while in others the condition went on to ankylosis, abscess formation *et cetera.* The reason, of course, was that both of these conditions were included in the diagnosis of tuberculosis of the hip.

The vertebral column has always offered examples of deformity in almost every conceivable shape, including the classical "angular curvature" and the very simplest of curves. Most of these before the advent of X rays were ascribed to Pott's disease, and possibly correctly so. Gradually, as radiography improved, other causes became apparent, such as congenital anomalies, hemivertebra *et cetera.* The residual paralyses following acute anterior poliomyelitis accounted for a good number. There still remained a number of cases unaccounted for by these causes, and many of these now are known to be results of osteochondritis.

Although the various conditions mentioned are included in the osteochondritides, it is not certain whether there is one common constant cause for the apparently numerous widespread manifestations of one pathological entity, or whether there may be several ætiological factors varying with the site of the lesion.

It is, however, interesting to note how opinion has changed in the course of a few years. In 1926 R. Lawford Knaggs, in his "Inflammatory and Toxic Diseases of the Bone", referring to osteochondritis, stated that:

> It is not necessary to assume an obscure interference with the osteogenetic function. The characteristic X ray appearances are evidently caused by a non-suppurative and destructive inflammation of bone, and the micro-organism responsible is almost certainly a staphylococcus of attenuated virulence. The affection is already familiar under the title "Serous or Albuminous Osteomyelitis".

This opinion was based on the growth of staphylococci on cultures taken from affected femoral heads and examination of material recovered at the same operations. These results have not been consistently obtained, however.

In January, 1933, Joseph Buchman in an article in *The Journal of Bone and Joint Surgery* wrote:

> Since 1888 a number of writers have described a group of conditions all of which have more recently been grouped under the generic term of osteochondritis, signifying a non-inflammatory derangement of the normal process of bony growth occurring at the various centres of ossification at the time of their greatest developmental activity. . . .

He ascribes the pathological changes found to stress and strain (that is, trauma) during this period of rapid growth, and this theory as to ætiology appears to be generally accepted. Endocrine dyscrasia has been put forward as a possible cause, but according to Buchman in the article quoted there is nothing to support the idea. Nevertheless, there is something suggesting support for this theory in the histories of many of these cases, and a very guarded prognosis should be given in children, particularly girls approaching puberty and the subjects of mild scoliosis. Not infrequently at about this time, when indeed one would expect the endocrine balance to be easily disturbed, there has been noted a very rapid change for the worse. The vertebral column at times appears almost to collapse, so rapid is the development of a major deformity. This phenomenon is not easily accounted for by stress and strain which are normal conditions and which do not materially alter.

A significant case was that of a girl, aged nineteen years, who had a thyreoidectomy performed (some time before I saw her). Her history was that she had always been round shouldered, but that after the operation she became very rapidly worse.

The removal of part of the thyreoid surely might cause some disturbance of endocrine balance.

Against the theory of trauma as the chief factor in ætiology is the fact that many cases of osteochondritis, particularly in the tarsal scaphoid, undergo spontaneous cure without any rest from normal activity. On the other hand, in Perthes's disease of the hip gross deformity will occur at times, even without weight bearing.

It is this very characteristic of indifference, one might almost say, to treatment which makes the condition, particularly in hip and spine,

so hard to manage, and which renders the giving of a prognosis so difficult.

When the spine is affected, the vertebral epiphyses in the X ray picture show irregularities and fragmentation, and the bodies exhibit a moth-eaten appearance during the acute stage.

The patient complains of pain and tenderness on pressure over the affected parts. Curvature, if not already present, soon makes itself obvious, and if it is not corrected and if the correction is not held, the deformity will increase and become fixed. There can be no question but that the strain of weight-bearing is bad and likely to increase deformity during this active stage.

Treatment is difficult, and we are definitely hampered by lack of knowledge.

Apart from general measures the important points are: (i) To prevent deformity as far as possible by removing strain; (ii) if deformity has already occurred, to correct it; and (iii) to hold the correction until the X ray picture shows us a return to normal.

CASE HISTORIES.

CASE I.—A.T., a female, aged thirteen and a half years, was seen on January 21, 1934. She had always been backward at school. She could not make any progress. She had not menstruated. Twelve months previously her mother noticed that the child's back was bent.

On examination the child appeared much younger than her years, and was obviously subnormal. There was marked kyphosis in the dorso-lumbar region. An X ray examination was made, and Dr. Sear and Dr. Voss reported as follows:

"There is a very slight degree of scoliosis at the lumbo-dorsal region, the convexity being to the right. There is an exaggerated lumbar lordosis with some compensatory kyphosis in the lower thoracic region, and considerable deformity of the lower thoracic and upper lumbar vertebræ. The exact nature of the condition is uncertain, and it might be elucidated by further skiagrams of the skull and long bones, but it appears to be rather of the nature of an osteochondrodystrophy, though not typical in appearance." (See Figure I.)

Treatment adopted was recumbency for two months, and then a series of corrective plaster jackets in an attempt to improve the deformity. Finally a type of Taylor brace, which is still being worn, and corrective exercises were used.

The last skiagram, taken on March 16, 1935, shows a definite improvement in shape and condition of the vertebral bodies. (See Figure II.)

CASE II.—G.C., a male, aged twenty-seven years, complained of pain in the back and weakness of lower limbs. On examination he was seen to be a large man with the youthful appearance of a boy of eighteen years, his voice was unbroken, he had no growth of pubic hair, his penis and testes were of the infantile type.

On X ray examination (Figure III) he was at first reported on as having secondary deposits of newgrowth. He was jacketed and put at rest, and successive films showed gradual improvement in the condition of the vertebræ. The diagnosis of *osteochondritis deformans juvenilis* was arrived at in spite of his age, as the epiphyseal plates in at least one situation (iliac crests) were still evident and unfused.

Clinically he is much improved, and his symptoms have disappeared. His skiagrams have been most interesting, but unfortunately the films have been unsatisfactory for reproduction, and Figures III and IV give but a very poor idea of the conditions shown.

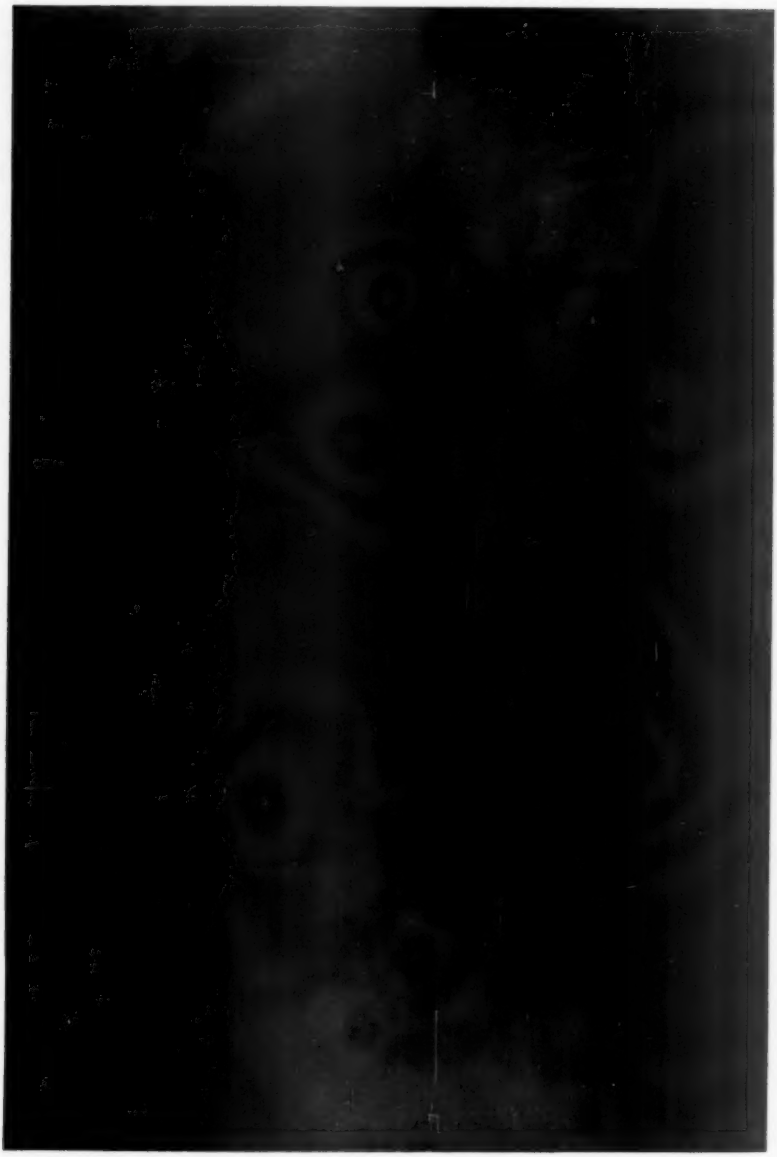

FIGURE I. *Osteochondritis deformans juvenilis*, showing the condition in Case I on May 11, 1934. The irregularity of the bodies of the vertebræ and of the epiphyses is well shown.

FIGURE II. *Osteochondritis deformans juvenilis*, showing the condition in Case I on March 13, 1935. Definite improvement in the form of vertebral bodies may be seen.

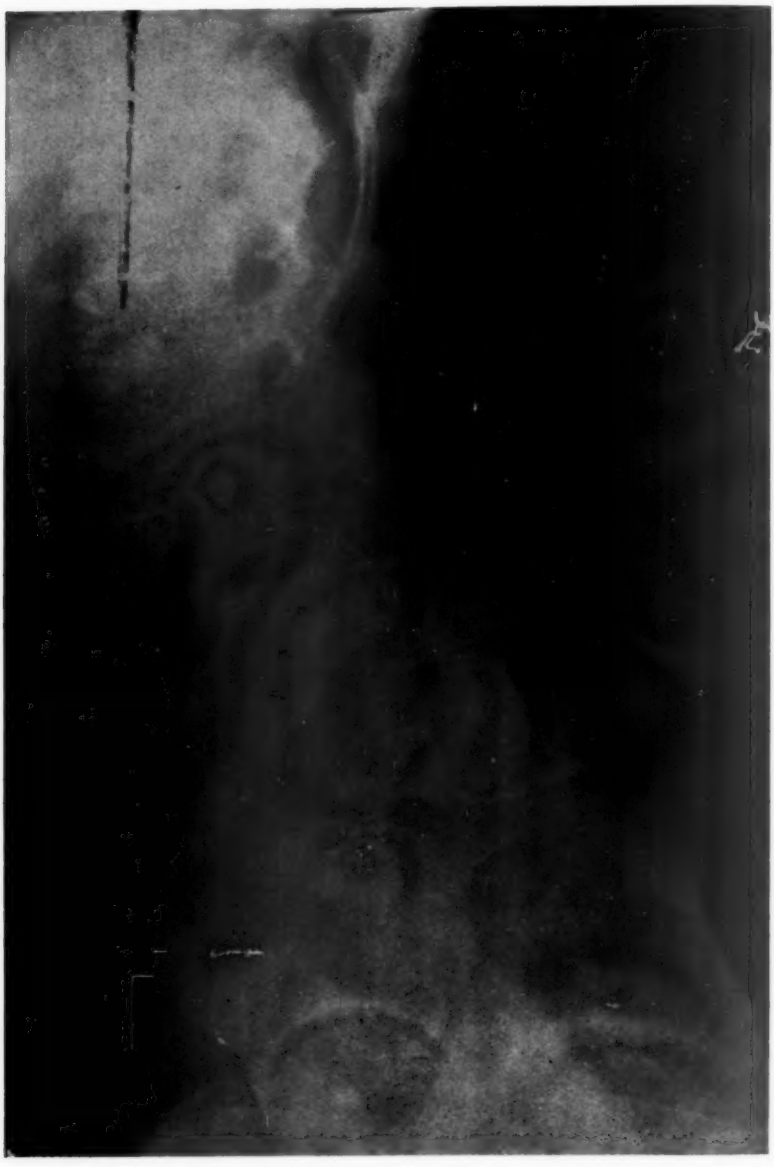

FIGURE III. *Osteochondritis deformans juvenilis*, showing the condition in Case II in September, 1934, lack of density of vertebral bodies causing poor definition. The marked irregularity of the bodies of the lumbar vertebræ can be well seen.

FIGURE IV. *Osteochondritis deformans juvenilis*, showing the condition in Case II in April, 1935. Note the improved condition of the bodies of the affected vertebræ, particularly lumbar IV.

SUMMARY.

It is suggested that endocrine imbalance cannot be dismissed as an ætiological factor in *osteochondritis deformans juvenilis*.

Trauma, although not a true ætiological factor, will produce and aggravate deformity during the acute stage.

Treatment in the present stage of our knowledge consists mainly in providing adequate protection from strain until X ray evidence shows the affected bone capable of performing normal function.

Two cases are quoted which appear to bear out the above suggestions.

Surgical Technique.

A METHOD OF EXTENDING A FRACTURED HUMERUS.

By R. Douglas Wright,
Surgeon to Out-Patients' Department, Melbourne Hospital;
Senior Lecturer in Pathology, University of Melbourne.

The question of extension to the humerus arises particularly in cases of compound fracture. In-patient hospital treatment is always necessary for cleaning of the wound and detection of infection in the first week or so. Immobilization of the fracture in the correct position is essential; the apparatus for immobilization must, however, allow inspection and dressing of the wound without the limb being disturbed; moreover, it must be so arranged that the position of the limb will not be disturbed when the patient moves longitudinally in the bed.

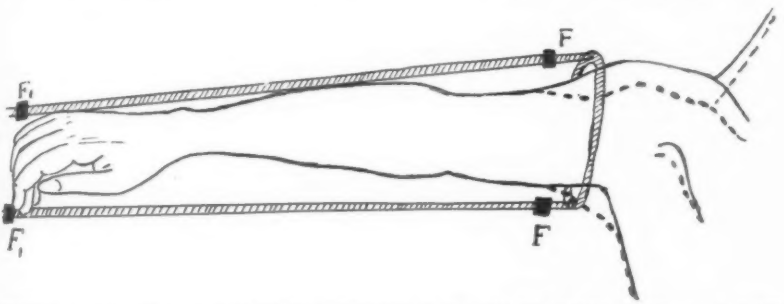

Figure I. The effect of longitudinal movement of the patient relative to the fixed splint is shown by the dotted line. The malposition produced at the fracture is demonstrated.

Longitudinal movement of a patient in a bed will certainly take place. Whether it be due to use of the bottle or the pan or just to restlessness, I have not yet observed a patient remain stationary in an ordinary bed. An apparatus such as Thomas's arm splint, being suspended by two points, does not allow this longitudinal movement, and when it occurs the arm is distorted in the splint (Figure I). Further, such appliances interfere very much with inspection and dressings. Another fault of these fixtures is that they put the elbow in full extension, which may interfere with the position of the fractured bone; and especially in supracondylar fractures they almost preclude the maintenance of the forward bend of the lower end of the humerus which is so essential for perfect function.

To overcome these difficulties I have tried a method of extension based on the principles of muscle rest enunciated by the late R. Hamilton Russell. A length of adhesive plaster is taken and firmly applied to the volar and dorsal surface of the forearm and a very free loop is left over the hand. A light

gauze bandage keeps the tape firmly adherent. Into this a spreader 7·5 centimetres (three inches) square is placed so that the dorsal and volar portions are equal in length. A pad of cotton wool sufficiently large to keep the wrist dorsiflexed is then placed in the palm of the hand. Cotton wool is smeared over the free area of the strapping to prevent adhesion to it of the back of the hand and the fingers. A cord is then attached at the centre of the spreader and passed over a pulley on an extension apparatus, so that the arm is approximately horizontal, a little less than right angle to the thoracic wall. The forearm is flexed to an angle of 135° with the humerus (Figure II).

When the patient is almost completely flat in bed the resting position of the arm is in abduction, as may be noted on the beaches, or by the frequency with which one has to request a patient on the examination couch to put the hands by

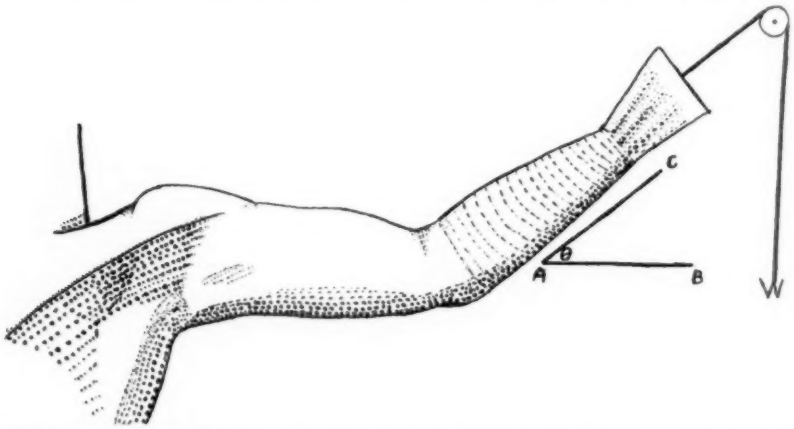

FIGURE II. Showing the general position of the humerus and forearm. Single point suspension is achieved. Longitudinal movement does not affect the position of the fracture. The extension in the line of the humerus is W cos θ. This may be supplemented along AB by strapping extension to the lower humerus or via a band round the proximal part of the forearm.

the side. The abduction of the arm is therefore not a stress on the patient, but a resting attitude. The almost flat position of the patient is essential for success with this suspension.

A weight of 3·1 or 3·6 kilograms (seven or eight pounds) provides sufficient extension to keep the arm free of the bed. The forearm swings into its position of rest in the mid-prone position, so avoiding over-stretching of the *pronator teres* muscle and valgoid deformity. The wrist is in dorsi-extension because of the pad of cotton wool in the palm. The humerus usually comes into good position or remains in good position after reduction.

Thus is produced extension of the humerus, forward bending of the humerus with the strap of triceps tendon supporting it on the dorsum, and the humerus is readily accessible for dressing and inspection as it swings freely.

As the upper extremity is suspended by the single strap to a single point, it does not become distorted by fairly large movements of the patient up or down the bed.

The method of extension may be used continuously throughout the treatment of the fracture. More usually when the wound is clean or healed some method of support suitable for the ambulatory patient is used.

PROCIDENTIA UTERI.

By F. A. MAGUIRE,
Sydney.

Anatomy.

In the Transactions of the Australasian Medical Congress (British Medical Association), Second Session, printed in the supplement to *The Medical Journal of Australia*, March 29, 1924, there is printed a paper by the author on the anatomy of *procidentia uteri*. It is based on a dissection of a specimen at the University of Sydney. The following summary and conclusions were arrived at:

1. In procidentia the protruding mass consists of a sac containing the bladder and uterus, together with their vascular and nervous supply, ligaments and ducts. The wall of the sac is the extroverted vagina. The sac is lined with peritoneum.

2. The bladder and uterus are below a line drawn from the inferior border of the *symphysis pubis* to the tip of the coccyx. The urethral orifice is on the same level as the top of the bladder.

3. The ureter is lengthened and its distal end is outside the vulvar ring for a length of thirty-five millimetres.

4. The bladder is stripped off the anterior wall of the uterus.

5. The rectum does not enter into the procidentia because it is slung to the anterior aspect of the sacrum by a series of obliquely placed fibrous septa.

6. The anal canal is displaced dorsally for about thirty-seven millimetres and lies further from the pubes instead of in its normal position about midway between pubes and coccyx.

7. The neuro-vascular bundles are greatly elongated, but retain their attachments to the pelvic wall and viscera.

8. The urogenital diaphragm is displaced downwards, and comes to lie almost vertically below the inferior rami of the pubes.

9. The *levatores ani* undergo several changes: (*a*) they retain their attachments to the pelvic walls; (*b*) they are greatly stretched and elongated; (*c*) throughout the greater part of their extent they are thin and membranous, the muscle tissue having atrophied; (*d*) their inferior borders remain thick and strong; (*e*) their medial margins are widely separated, but retain their attachments to the lateral vaginal wall; (*f*) their junction behind the rectum is displaced dorsally and comes to lie almost vertically below the tip of the coccyx; (*g*) the hiatus between the medial margins of the muscles is thus greatly widened and lengthened—in the normal pelvis its length is seventy-two millimetres and its width twenty millimetres, in procidentia its length is one hundred and seven millimetres, and its width fifty-four millimetres.

10. As a result of 8 and 9 the pelvic floor is opened and the viscera—uterus, vagina and bladder—fall through the gap.

11. The pelvic floor as a whole is at the same time displaced downwards.

The surgeon must bear these facts in mind when approaching a case of *procidentia uteri*, for it is only by a reconstruction of the displaced parts that a successful result can be looked for. In women who are past the child-bearing age, or in those in whom an operation for sterilization is performed or has been performed, every case of *procidentia uteri* can be completely cured by an operation undertaken from below.

The author has had personal experience of 119 cases of total prolapse.

Age Incidence.

The age at which each patient reports for examination and treatment is far from being the age at which the total prolapse first appeared, and still further from the time when the first symptoms appeared. The ages at which patients in this series reported were as follows, namely: the youngest was twenty-five years of age, the eldest was seventy-two years. The groups under which they came were as follows: 20 to 30, 1; 31 to 35, 2; 36 to 40, 7; 41 to 45, 15; 46 to 50, 18; 51 to 55, 19; 56 to 60, 18; 61 to 65, 23; 66 to 70, 11; over 70, 3.

Many of these patients had had a total prolapse for periods varying from two to twenty years before reporting for operation. The first symptoms, however, appeared in many patients long before a total prolapse occurred, namely: 15 to 20, 3; 21 to 25, 8; 26 to 30, 10; 31 to 35, 15; 36 to 40, 8; 41 to 45, 14; 46 to 50, 9; 51 to 55, 5; 56 to 60, 5; 61 to 65, 4; 66 to 70, 2; over 70, 1.

In the majority of cases the first symptoms appeared long before the menopause, but the majority of the patients reported for operation after the menopause.

In 53 cases in which the information is available, the average time between the appearance of the first symptoms and the time of reporting for treatment is as follows: 1 to 5 years, 29; 6 to 10 years, 11; 11 to 15 years, 16; 16 to 20 years, 8; 21 to 25 years, 11; 26 to 30 years, 2; 31 to 35 years, 1; 36 to 40 years, 4. From this it will be seen that 18 patients allowed the condition to progress for over twenty years; 24 patients allowed it to progress from ten to twenty years, and 40 patients from one to ten years. The oldest patient in the series was seventy-two years of age. In this instance the total prolapse descended suddenly one month before she reported.

Ætiology.

At least 41 of the patients definitely stated that the first symptoms were associated with the birth of their children. In most cases the condition was associated with the birth of the first child. The commonest contributing cause to the occurrence of total prolapse is undoubtedly the period of the menopause. Eighty-seven patients reported at or after the period of the menopause. The average age of the menopause in the 119 patients reported on was forty-eight years. The table showing the age of reporting shows that only one patient reported under the age of thirty years and only one under thirty-five. There were seven between thirty-six and forty, and 15 between forty-one and forty-five. The remainder of the patients had a condition which was definitely associated with the menopause. It is a very common experience to find the patients state that they had a certain amount of discomfort or trouble following childbirth, but that at or about the period of the menopause the condition suddenly became very much worse. In a few cases definite strain is associated with the occurrence of the total prolapse, as in one patient the total prolapse was associated with a severe attack of vomiting during an attack of gall-bladder inflammation; another was definitely associated with a severe attack of asthma; while in a third it was directly due to straining while attempting to lift an overturned motor car from an imprisoned child. In another case the patient blamed climbing stairs after moving into a two-storied house. All these are of course factors which increase the intraabdominal tension.

In one case in which the patient had never been pregnant a neuro-muscular disturbance was suspected. An X-ray examination revealed an occult *spina bifida* extending from the lumbar to sacral vertebræ.

Number of Children.

In 84 patients of the series the number of children were as follows, namely: 0, 2; 1, 8; 2, 17; 3, 19; 4, 17; 5, 8; 6, 12; 7, 8; 8, 6; 9, 3; 10, 2; 11, 1; 12, 1; 13, 1.

About 78 had had three children or more, while 42 had had five children or more.

Symptoms.

In the majority of cases the patients complained mainly of a mass protruding from the vulva, or as they put it a "lump" coming down between the thighs. Backache was complained of by only 41 patients and bearing down by only 32. Frequency of micturition was usually associated only with cases in which there was some degree of cystitis, while incontinence was present in seven of the series. Forty-three patients complained that they had to push the mass up before they could pass urine.

Physical Signs.

Examination showed the usual appearance of a procidentia with the cervix at the apex of an everted vagina. In 54 patients of the series, ulceration to a lesser or greater degree was present on the cervix. In one instance only was carcinoma found on the cervix associated with procidentia. Only in cases in which there was ulceration, was there a history of vaginal discharge as well.

Menstruation.

In 38 patients menstruation ceased at the average age of forty-eight years. Thirty-two of the patients were still menstruating when they reported for treatment. In 30 cases there was no record of the menstrual history.

Treatment.

Treatment in general was either by support in the form of a rubber semi-fluid ring or by operation.

No treatment was given in seven cases. One patient was too fat for operation and was sent away to reduce; she did not return. One patient complained of gall-bladder trouble, and although she had a total prolapse, she was not complaining of it and refused to have anything done for it. One patient refused operation or treatment of any kind. One patient had a *spina bifida occulta*; it was not considered wise to operate on her; she was fitted with a stem support at a later date. One died under the anæsthetic before operation was performed. One refused operation; she is wearing a stem pessary.

A support in the form of a ring has been worn by eight patients of the series. The patients are as follows: Number 1, aged sixty-six years, a very poor subject for operation; Number 2, aged fifty-four years, had a systolic blood pressure of over 200 millimetres of mercury; Number 3, aged seventy-two years, suffered from severe bronchitis and was a very bad operative risk; Number 13, aged sixty-two years, had a systolic blood pressure of over 200 millimetres of mercury; Number 14, aged sixty-seven years, suffered from chronic pyelitis and declined operation; Number 26, aged 69 years, had a systolic blood pressure of just on 200 millimetres of mercury; Number 29, aged seventy-two years, was a very poor operative risk; Number 56, aged sixty-five years, had a very poor cardiac condition and was considered a very bad operative risk.

All these patients, with the exception of the last, had been wearing a ring from size 9 to size 12. The last patient could not wear a ring, and an Aveling support is being worn by her.

Eighty-two patients in the series have been treated by plastic operation performed from below. Eight of the 63 had in addition a double salpingectomy and ventrofixation. Four patients only were treated by vaginal hysterectomy with a subsequent repair of the pelvic floor. One patient who had carcinoma of the cervix, was treated by a radical abdominal hysterectomy with restoration of the pelvic floor. One had a total hysterectomy for fibroids and a repair of the pelvic floor, while two patients had a pyometra which was drained. Each then had vaginal hysterectomy.

There have been two deaths in the series. One patient who has previously been mentioned died under anæsthesia before the operation was performed, and another patient who had had three previous operations died from uræmia and pelvic peritonitis from the separation of adhesions in the pouch of Douglas.

Post mortem examination showed that the ureters were not damaged during the operation. Eight patients of the series are still to be operated on. The type of operation that has been performed in these cases is one that has been evolved by the author. The operation will now be described.

The Operation for the Cure of Procidentia Uteri.

It is wise to keep a patient with total prolapse in bed for at least one week before operation. During this time the vagina should be douched twice a day and firmly packed with gauze to hold the uterus at its proper level. This treatment will reduce the congestion and œdema in the vaginal wall, and give ulcers a chance to heal. The blood loss at operation is reduced, and the operation is more easily performed.

The patient is put in the lithotomy position and a catheter is passed. An Auvard's speculum is inserted and the cervix is seized by a vulsellum applied to the anterior lip. The uterus is examined and curetted in the usual way. After a sound has been passed, the cervical canal should be dilated up to at least 9 to 12 graduated dilators, and the cavity of the uterus thoroughly curetted. If nothing abnormal is found, the operation may be proceeded with. (If the uterus is found to be very unhealthy, it may be preferable to do a vaginal hysterectomy as part of the operation).

The uterus is then painted out with tincture of iodine and a strip of iodoform gauze 2·5 centimetres (one inch) wide is passed right up to the fundus of the uterus. The gauze is left long and is carried out on to the handle of the vulsellum. A second vulsellum is now applied to the posterior lip, and the cervix with the vagina is drawn down as far as possible. The whole of the field is then painted with spirit.

A Moynihan's bile-duct clip is then placed on the vaginal wall at a spot which is selected by the surgeon. This point should be the point which will come together with its fellow of the opposite side after the cervix has been amputated and the anterior vaginal flap removed. It should be at least half-way between the front and back of the vaginal wall—for preference a little behind half-way—and at least 2·5 to 3·75 centimetres (an inch to an inch and a half) from the external os. These forceps are held by assistants at tension, and the cervix is strongly drawn down by the vulsella. A crescentic incision is made from the clip on one side down towards the cervix and up to the clip on the other side. It will lie about 1·25 centimetres (half an inch) behind the external os as a general thing at its lowest part. The cut is carried through the vaginal wall. At this point it is below the reflection of the bladder. If there is any doubt in the mind of the surgeon as to where the lowest point of the bladder is, a uterine sound can be passed through the urethra into the lowest part of the bladder to indicate to the surgeon the level where the lowest point (which is really the highest point) of the bladder lies. (See Figure I.)

The upper edge of the incision is now picked up about three millimetres on either side of the mid-line by two pairs of artery forceps. The edge is then lifted up. With the point of the scalpel the surgeon endeavours to find the plane between the vaginal wall and the vesico-vaginal septum. The point of a Mayo's scissors curved on the flat is then inserted between the vaginal wall and the vesico-vaginal septum and gently pushed up towards the urethra for a distance of about 3·75 centimetres (an inch and a half). If the scissors are gently opened and closed in their progress they will open up this plane. This is shown in the small inset (*a*) in Figure II. The scissors are then withdrawn and used to cut through the vaginal wall superficial to the plane of separation (inset *b* in Figure II). The vaginal wall is then picked up on either side at the highest point of the cut margin. (See Figure II.)

The process is repeated. The blade of the scissors is passed in for a further 2·5 to 3·75 centimetres (inch to an inch and a half), and the cut is made through the vaginal wall in the same way. On separating the edges, the vesico-vaginal septum should show up between the margins of the cut. If, however, the point of the scissors has gone too deeply (and it is very easy to get too

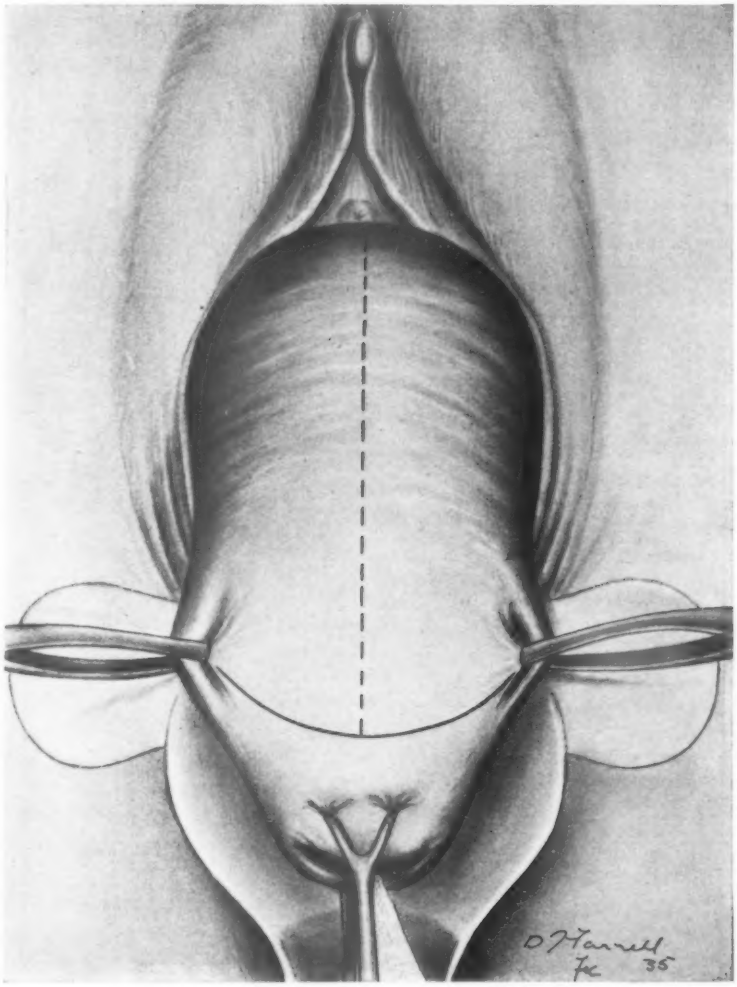

FIGURE 1.

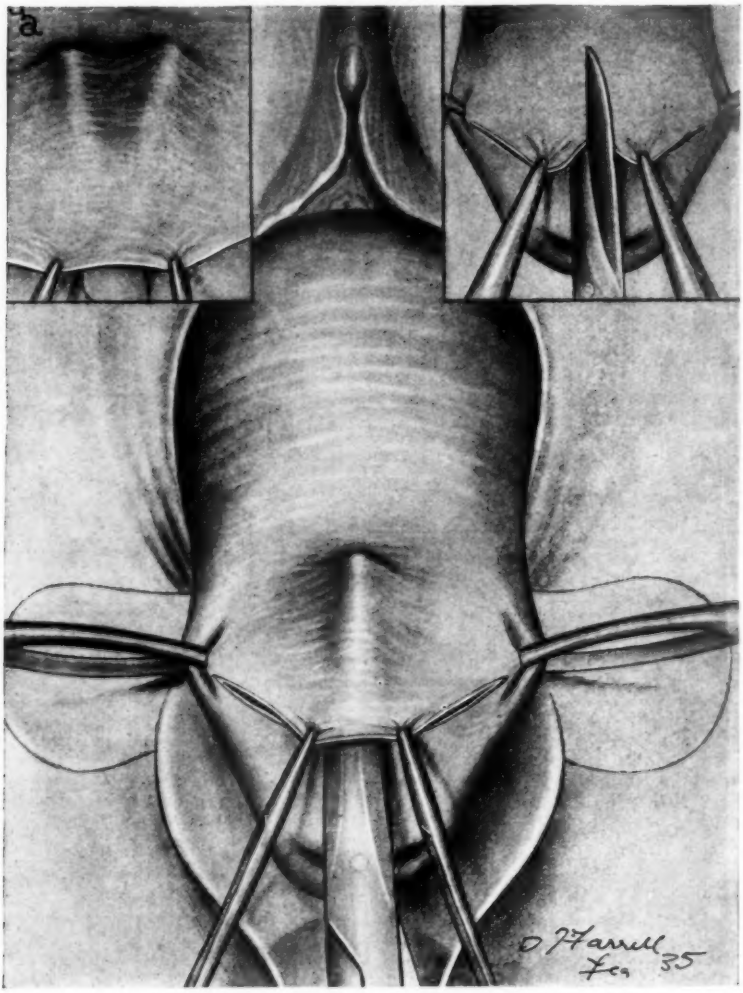

FIGURE II.

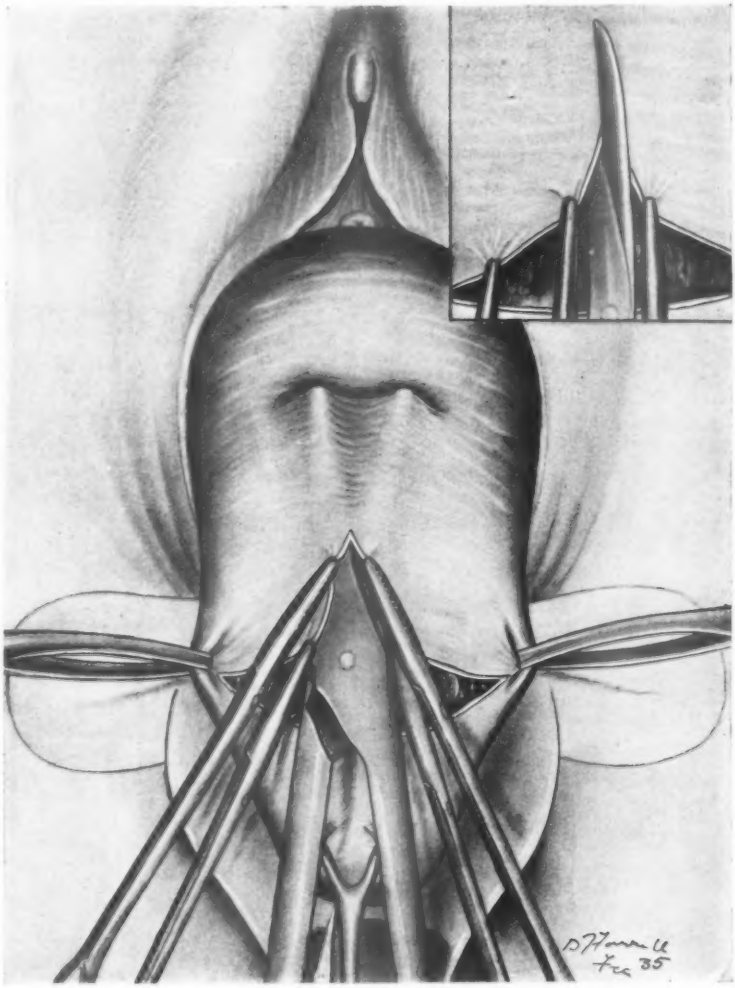

FIGURE III.

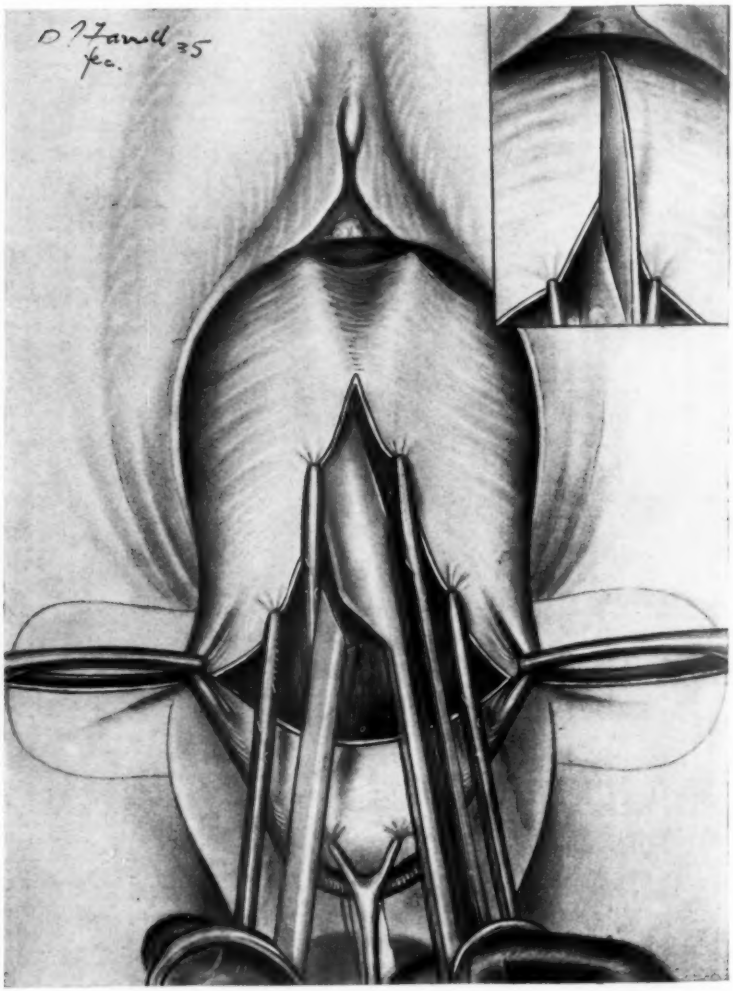

FIGURE IV.

FIGURE V.

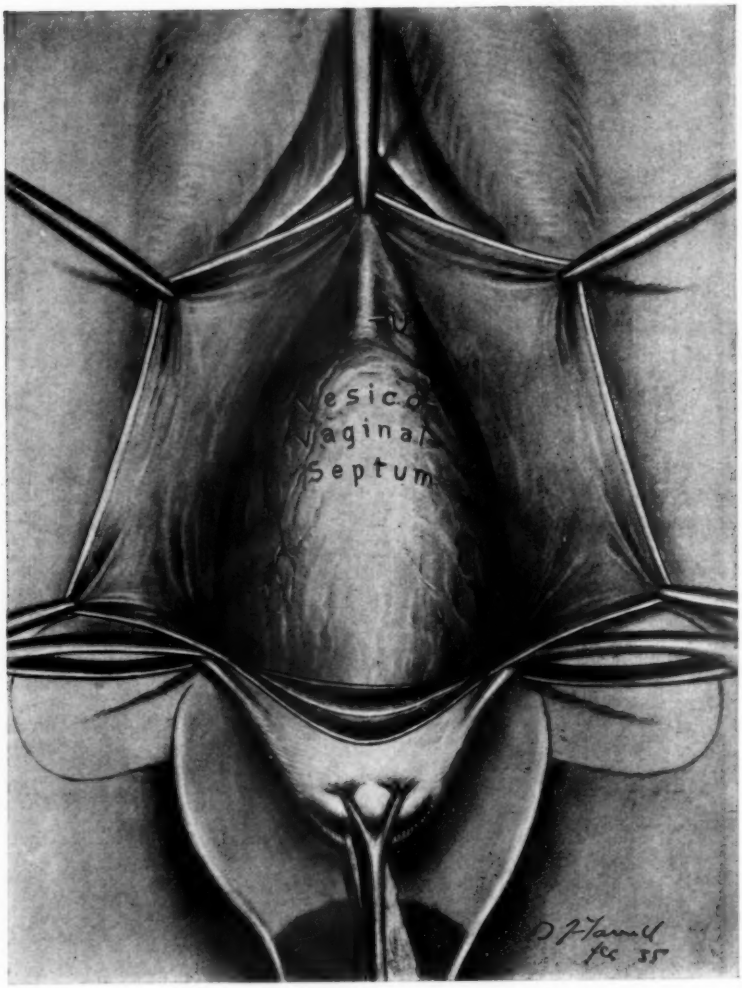

FIGURE VI.

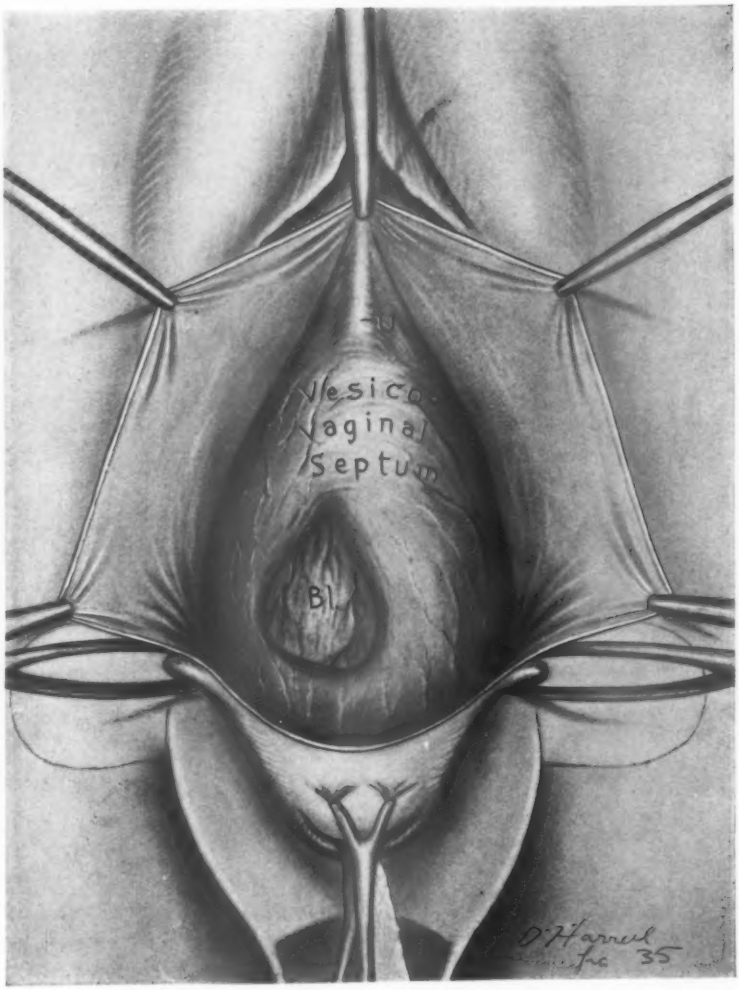

FIGURE VII.

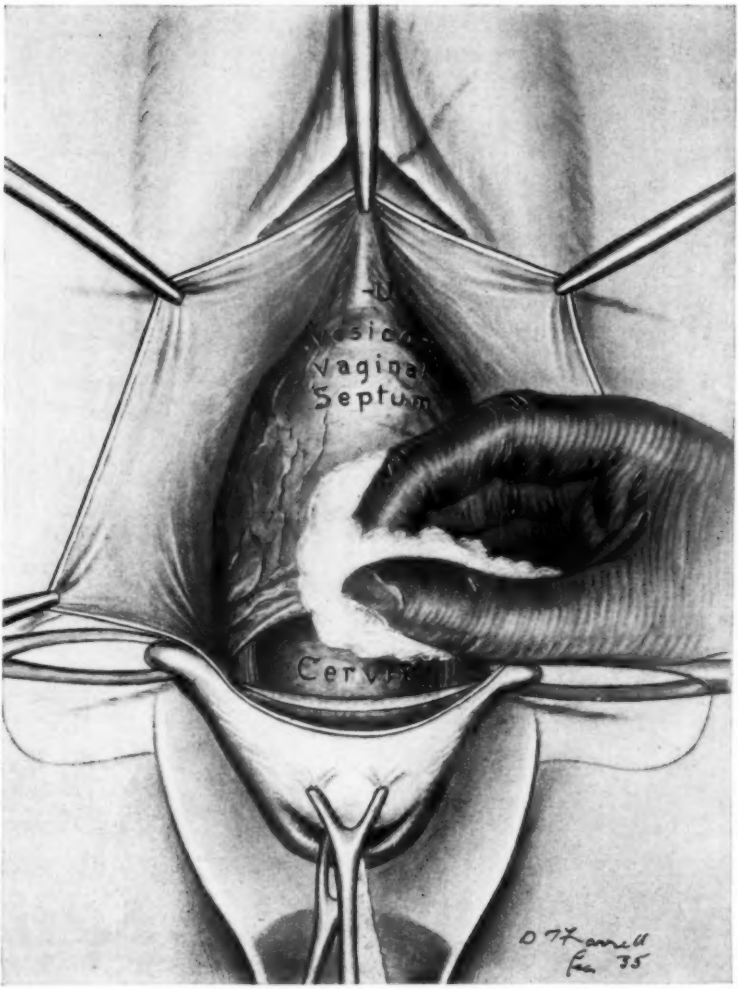

FIGURE VIII.

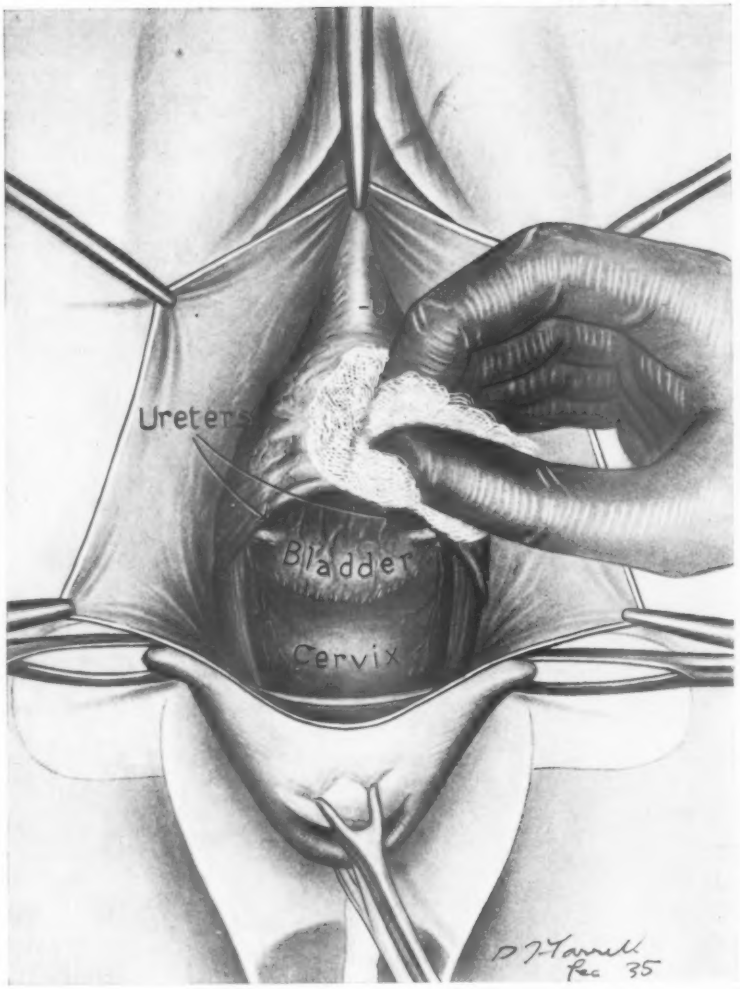

FIGURE IX.

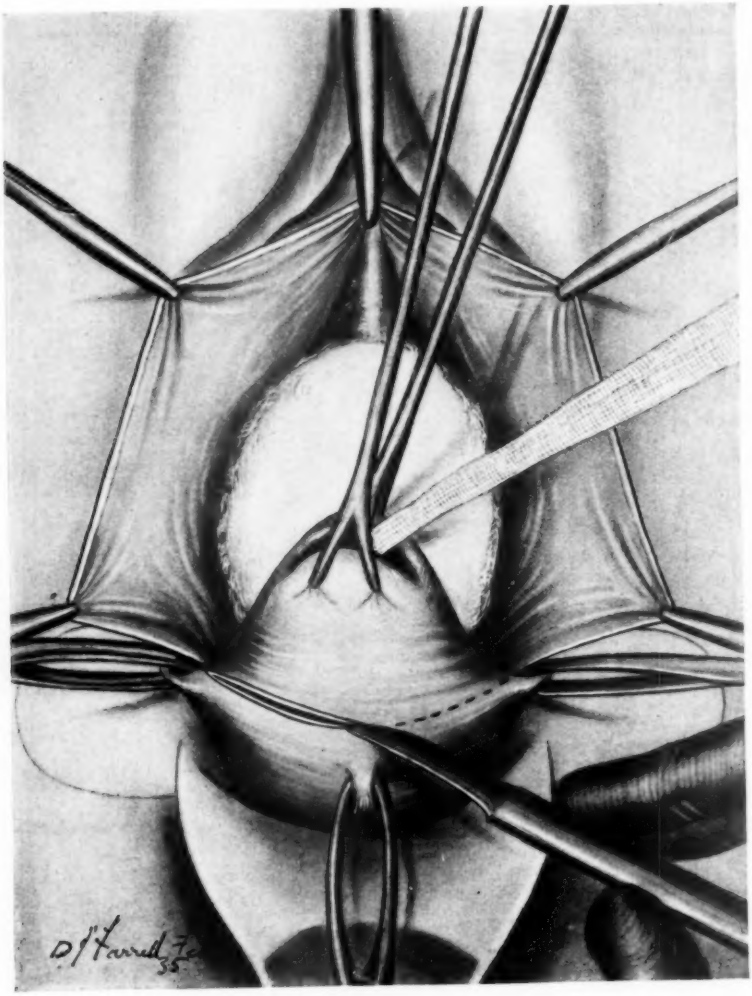

FIGURE X.

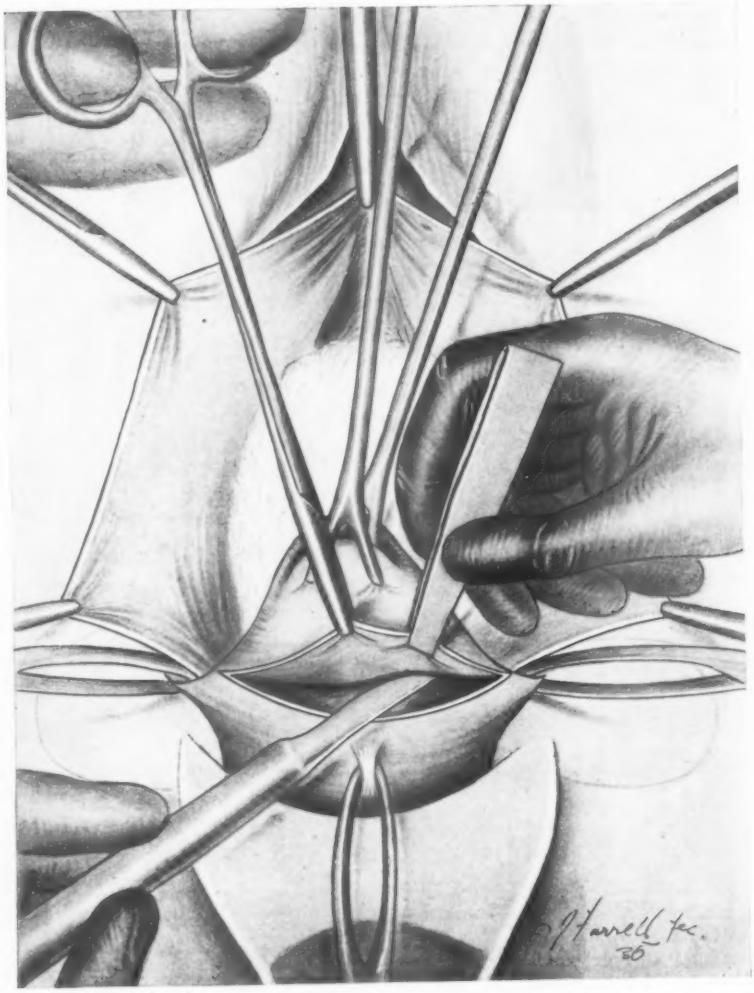

FIGURE XI.

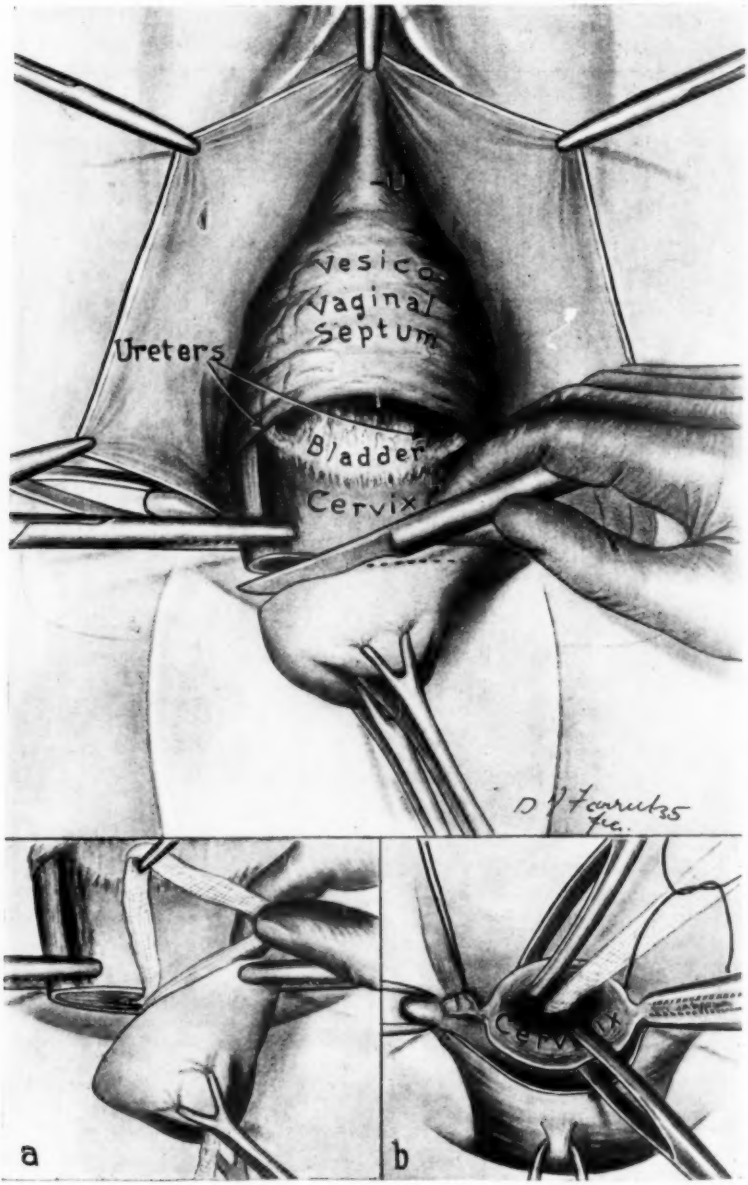

FIGURE XII.

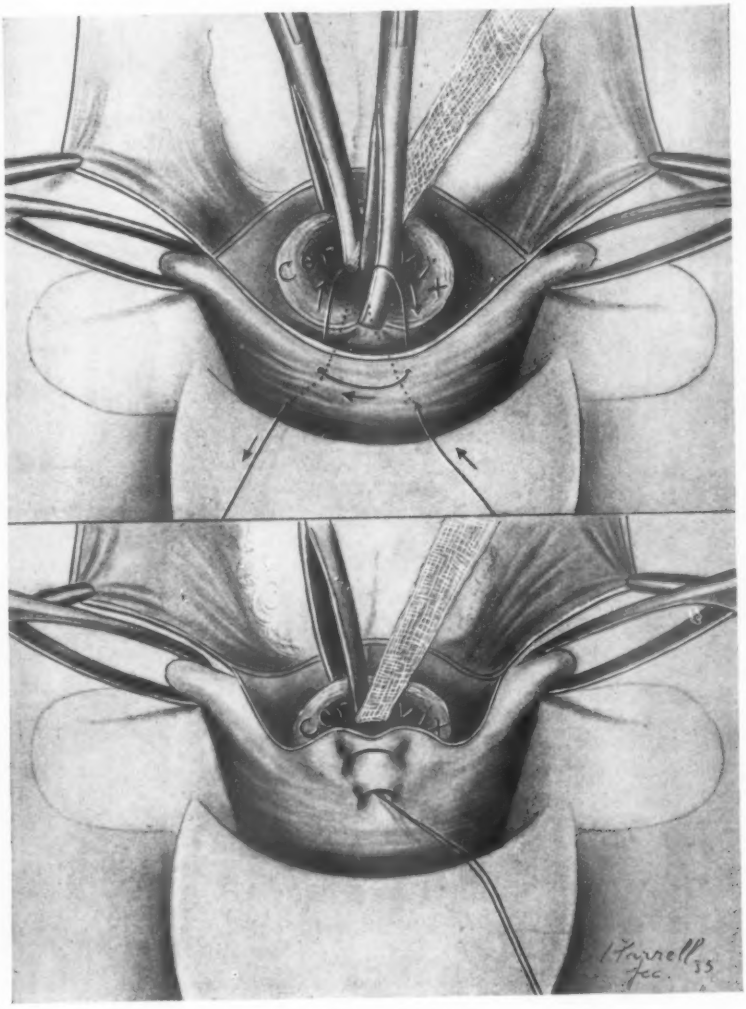

FIGURE XIII.

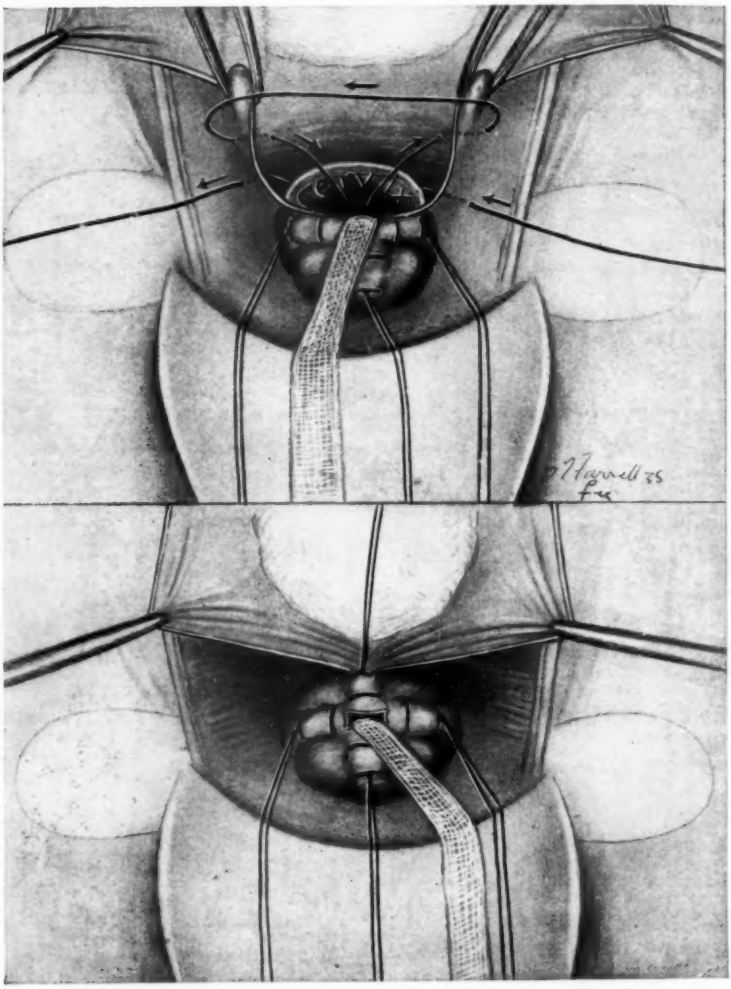

FIGURE XIV.

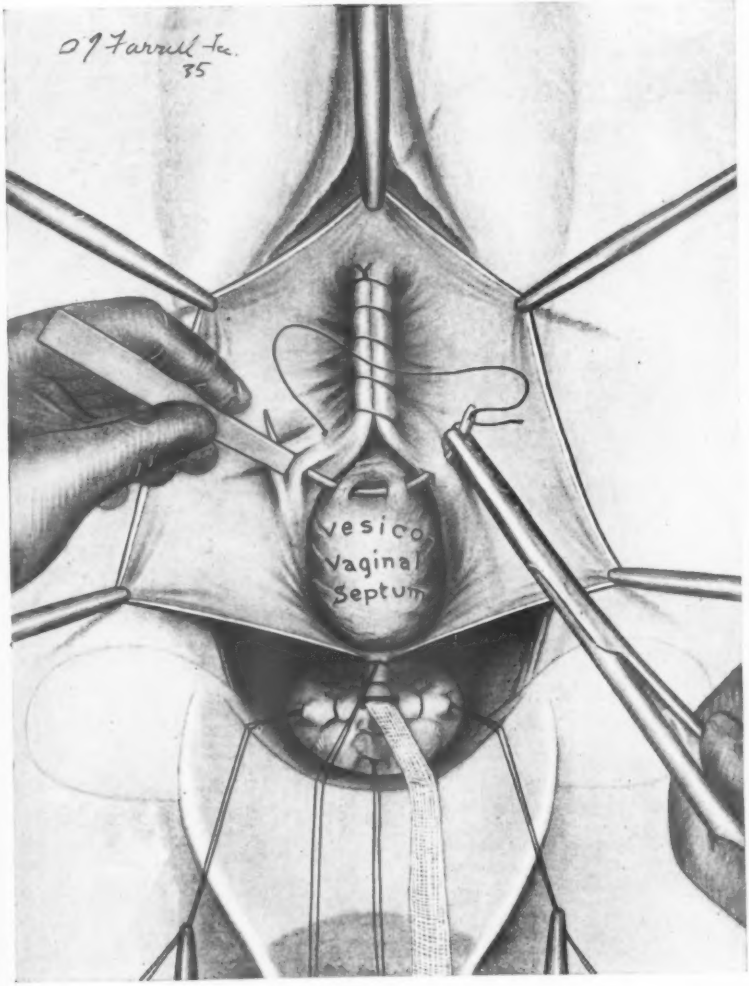

FIGURE XV.

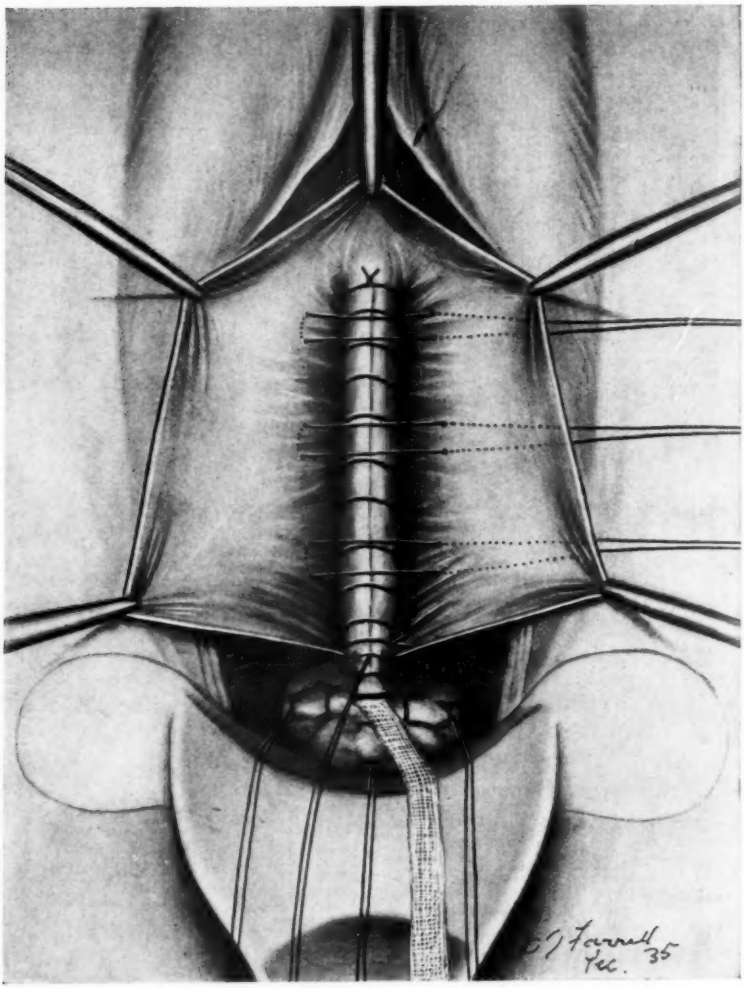

FIGURE XVI.

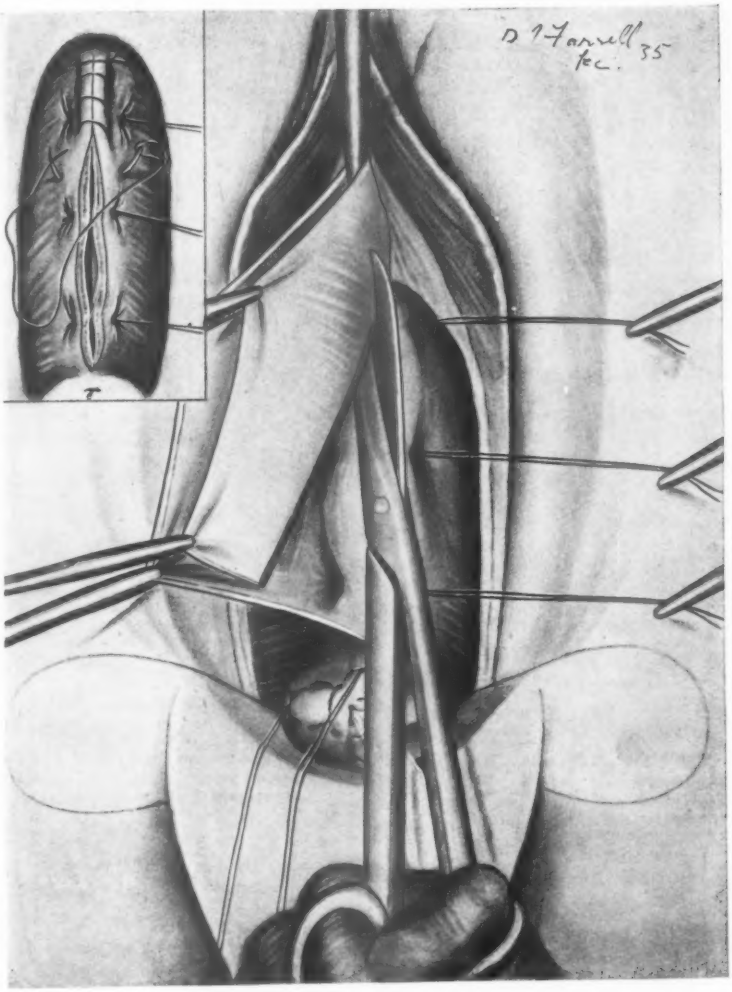

FIGURE XVII.

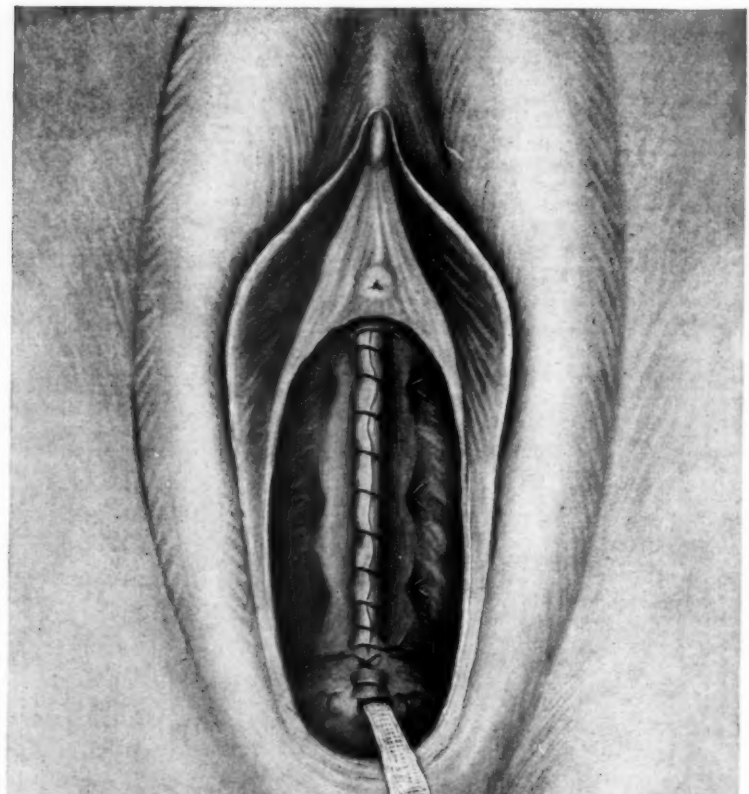

FIGURE XVIII.

deep if the prolapse is old standing and the tissues are tough and adherent), an examination of the cut edge will show a very definite double layer, the more superficial one being the vaginal wall, and the second one being the vesico-vaginal septum. The tissue underlying the cut in the mid-line will then be the wall of the bladder. The surgeon should endeavour before going on with the next stage to place the point of the scissors definitely in the plane between the vesico-vaginal septum and the vaginal wall. (See Figure III.)

The process is continued until a point is reached 1·25 centimetres (half an inch) below the urethral orifice. In cases in which there is a marked urethrocele associated with the cystocele, the lower part of the separation must be done very carefully and accurately, or the underlying urethra will be quite easily injured. When the incision is carried up to a point 1·25 centimetres (half an inch) below the urethra, a Kocher's forceps is put on the vaginal wall at the highest point of the incision. This is held by an assistant and drawn upwards. If the incision is carried too far up at this stage, troublesome bleeding is likely to be encountered from the urethral venous plexuses. It is likely to give the surgeon considerable trouble unless it is dealt with at the time by fine plain gut stitches inserted under the bleeding sinuses. (See Figure IV.)

The forceps attached to the left hand margin of the cut, that is, the edge on the surgeon's right hand, are drawn in tension. The Kocher's forceps at the upper end of the incision is drawn up, while the vulsella on the cervix are drawn down. A few gentle touches with the scalpel, as shown in inset *a* in Figure V, will free the vesico-vaginal septum, and if the vaginal wall is kept at proper tension by the assistant "fanning" out the forceps which are attached to the margin, a gauze swab held in the hand will easily detach the vesico-vaginal septum from the deeper layer of the vaginal wall. This is carried out until the point is reached where the bile-duct clip is attached to the vaginal wall. The line is made straight from the upper end of the incision to the clips on the lateral margin of the vaginal wall which the surgeon has taken for his lateral landmark. When this reflection is completed, there are usually three or four bleeding points to be tied off. If a sponge well wrung out in hot saline solution is firmly applied to the raw surface for half a minute, it will stop the greater part of the oozing, and not more than four or five bleeding points should need to be tied. These are tied with fine gut before the surgeon proceeds to the next stage. This should leave a dry field. (See Figure V.)

The same steps are repeated on the opposite side, so that the vaginal wall is turned back as a triangular flap on either side of the mid-line. The apex is 1·25 centimetres (half an inch) below the urethra, and the base extends from the bile-duct clip which is the lateral landmark, out to the artery forceps which were originally placed three millimetres from the mid-line. This is illustrated in Figure V.

Figure VI shows the complete lateral reflection of the vaginal wall with exposure of the vesico-vaginal septum. Just below the point of the Kocher forceps at the upper end of the incision, the tubular urethra can be seen. The vesico-vaginal septum passes downwards towards the cervix (this is really upwards when the cervix is in its proper position in the body, but with the complete prolapse it is pointing downwards towards the cervix, which is now the lowest part of the vagina). At this point the vesico-vaginal septum is firmly attached to the cervix and forms a complete cover for the bladder which lies underneath. In order that the bladder may be pushed up and freed from the cervix, it is necessary to cut through the attachment of the vesico-vaginal septum to the cervix. The incision by which this is done is shown in Figure VI about three millimetres above the level of the incision in the vaginal mucous membrane.

Figure VII displays the condition of things which occurs if the incision through the vaginal wall is carried through the vesico-vaginal septum. An opening is shown in the latter structure and through it the bladder wall is displayed. This occurs not infrequently. As long as it is recognized by the surgeon and repaired no harm results.

The vesico-vaginal septum is now separated from its attachment to the cervix (Figure VIII). After the incision has been made with the scalpel, the

margin of the vesico-vaginal septum can be rolled back with a piece of gauze held by the surgeon in the finger and thumb of the right hand, while he makes traction on the vulsella on the cervix. The anterior surface of the supravaginal portion of the *cervix uteri* now comes into view. The reflection is carried back quite easily in the mid-line, but when the surgeon approaches the lateral angle of the incision, some bleeding may be encountered owing to the fact that the cervical branches of the uterine arteries lie in the connective tissue in the plane below or deep to the vesico-vaginal septum.

In Figure VIII the cervix itself is shown displayed by the surgeon in the mid-line, while at the lateral margin of the cervix the pelvic connective tissue, which is given by some the special name of the lateral cervical ligaments or the ligaments of Mackenrodt, is shown. This pelvic connective tissue is really the condensed connective tissue associated with the uterine vessels and their branches and with the ureter.

The reflection of the vesico-vaginal septum is carried upwards in the mid-line until the bladder is displayed (Figure IX). The bladder strips easily off the anterior surface of the cervix. The reflection is carried out on either side until the ureters can be seen passing from the lateral angle of the bladder around the lateral margin of the cervix. It will be remembered that the ureters approach the bladder lateral to the cervix. They vary in the distance at which they lie from the cervix. They may be from five to twenty-five millimetres lateral to the cervix, but usually in total prolapses they are under so much tension that they lie close to the cervix on either side. The reflection should be carried up on either side until the ureters are definitely displayed. Unless this is done there is grave danger of their being caught up by sutures at a later stage. At the same time the lateral pelvic connective tissue with the contained uterine vessels and their cervical branches is displayed lateral to the cervix. Considerable bleeding may be encountered at this stage at the lateral angle of the incision. This is due to the fact that the cervical vessels may be enlarged on account of hypertrophy and inflammation of the cervix. The bigger the cervix—the more hypertrophied and ulcerated it has been—the more will there be bleeding at the lateral angle of the incision. When the reflection of the vesico-vaginal septum and the bladder is completed, it should be possible to push the bladder with a swab almost out of sight between the margins of the vagina and the front of the cervix. Bleeding vessels are always encountered along the margin of the vesico-vaginal septum, mainly in the mid-line. These are branches of the azygos vaginal arteries. Thorough hæmostasis should be established at this stage before anything further is done. This completes the reflection on the anterior surface of the cervix. The surgeon is now ready to proceed to the next stage; this is shown in Figure X.

A gauze pack is placed over the bladder, and it is held up in position by an assistant. The vulsella attached to the cervix are now carried upwards and forwards, and tension is placed on the lateral clips. A third clip is now placed on a point selected by the surgeon fairly high up on the posterior vaginal wall. This point will ultimately become the posterior edge of the cervix after amputation. An incision is now made from one lateral clip across to the other, and up to the point where the clip is placed on the vagina in the mid-line. This incision is carried through the vaginal wall only. The vaginal wall is now reflected downwards, that is, towards the cervix, for about 1·25 centimetres (half an inch), so as to expose the underlying cervix. If the pouch of Douglas is very low, it may be opened at this stage, but it is easily recognized by the shining peritoneum which lines it. It does not matter if it is opened, but it should be closed at once with a fine catgut stitch. The cervix is now entirely freed of vaginal wall, front and back.

Figure XI shows the reflection of the vaginal mucosa from the cervix posteriorly. It is carried out on either side to the clip and is entirely free.

Figure XII, with its insets *a* and *b*, shows the amputation of the cervix. Inset *a* shows the lateral pelvic connective tissue to the side of the cervix seized with Kocher's forceps on the right side. The point at which the cervix is to be amputated must be judged by the surgeon in each case, but at least 2·5 centimetres

(an inch) in ordinary cases and possibly 5·0 centimetres (two inches) or more in cases of hypertrophy of the cervix, can be removed with safety. If the cervical endometrium appears unhealthy, the deeper parts of the cervix can be "coned out" by carrying the incision in the cervix a little higher with each sweep of the knife. (See Figure XIII.) The Kocher's forcep is put on the lateral side of the cervix at least six millimetres (a quarter of an inch) below the ureter in order that the stitch that is placed above it may not damage or include the ureter. It is this step which shows how essential it is to expose the ureter on each side. The Kocher's forceps having been placed in position, the cervix is then amputated and the margin of the cervix is grasped anteriorly and posteriorly with two more forceps, as shown in inset *b* in Figure XII. A stout plain catgut stitch is now inserted behind each Kocher's forcep and tied. The ends are left long. This loop of gut is attached to the lateral pelvic connective tissue, and it may be carried forward to the front line in front of the cervix and stitched there below the bladder to give additional support to the bladder and cervix. The exposed cervix must now be reconstituted by bringing the reflected margins of the vaginal mucosa over it. The point of vaginal mucosa on each lateral aspect must be brought round to the front of the cervix, and the vaginal mucosa behind the clips is used to cover the raw surface of the cervix. This is done by the insertion of four Sturmdorff stitches: one in the mid-line posteriorly, one on either side, and one anteriorly. The details of the insertion of the posterior Sturmdorff stitches are shown in Figure XIII.

The needle with strong chromicized gut (at least number 2 chromicized, possibly number 3) is inserted at a point at least 2·5 centimetres (an inch) from the margin of the mucosa and 1·25 centimetres (half an inch) to one side of the mid-line. It is carried through the vaginal mucosa and right through the posterior lip of the cervix to emerge in the cervical canal behind the gauze which lies in the cavity of the cervical canal. It is then brought back and threaded through the vaginal mucosa about six millimetres (a quarter of an inch) from the margin from within outwards. It is carried across the mid-line and reinserted from without inwards. It now passes over the cervix to be inserted into the lumen of the cervical canal. It then passes through the posterior wall of the cervical canal and the posterior vaginal mucosa, emerging about 2·5 centimetres (an inch) from the cut margin and about 1·25 centimetres (half an inch) to the other side of the mid-line. When this stitch is drawn up and tied, it carries the vaginal mucosa over the edge of the cervix and reconstitutes the posterior lip. At the same time it draws the cervix definitely up towards the pelvic cavity. A similar stitch is now placed in either lateral angle of the cervix, and the lateral margins are covered. Chromicized gut is used at this stage, because it is on the surface, and the knot can come away after the chromicized gut is absorbed. It usually takes from three to six weeks to absorb and come away. If plain gut is used, it is absorbed too quickly, and secondary hæmorrhage is almost sure to occur about the end of the third week.

Figure XIV is rather an intricate one. It illustrates the insertion of the anterior Sturmdorff stitch. It is a very important stitch, as it brings everything anterior to the cervix together, and restores the cervix to its proper level. When it is drawn up and tied, it brings together the lateral pelvic connective tissue and the anterior vaginal wall, drawing together in the mid-line the points where the clips were on either side.

A continuous suture of plain number 2 gut on a curved round needle is now inserted, commencing at the Kocher's forceps below the urethra. (See Figure XV.) It bites into the deeper layer of the vaginal wall on either side, and picks up the vesico-vaginal septum on either side of the mid-line, bringing these structures together in the mid-line. This lifts the bladder up and supports it at its proper level by its normal supports, the lateral vaginal wall receiving an attachment through the triangular ligament to the inferior rami of the pubis and ischium. At the lowermost point this suture bites into the anterior surface of the cervix and completely restores the bladder to its proper level.

Figure XVI shows the insertion of three mattress sutures of number 2 chromicized gut to pick up the vaginal wall immediately superficial to the line

where the previous stitch has been placed. These three stitches are tied and clamped and give a line along which the redundant vaginal mucosa is removed in a triangular sheet.

The redundant vaginal mucosa beyond the line of the mattress sutures is removed with scissors. (See Figure XVII.) These are triangular sheets of mucosa. This leaves the cut margins of the anterior vaginal wall close together in the mid-line. They are united (see inset *a* in Figure XVII) by a continuous suture of number 2 chromicized gut.

The final result is shown in Figure XVIII, where it is seen that the vaginal wall is restored to the original length of 7·5 centimetres (three inches); the cervix is thrown back into the hollow of the sacrum and the patient now needs a thorough perineorrhapy[1] to complete the operation. It is my custom to stitch in a catheter at the end of the operation and leave it in for about six days. The bladder should be irrigated daily with a weak solution of permanganate of potash.

[1] *Vide* paper by the author: "A Simple but Rapid Perineorrhaphy", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, October, 1933.

Case Reports.

INTERNAL HERNIA THROUGH FORAMEN OF WINSLOW.

By J. K. DAVIDSON,
Assistant Surgeon, Christchurch Hospital, New Zealand.

INTERNAL hernia through the foramen of Winslow is a condition of great rarity, and from a perusal of the literature the number of cases reported up to the present number 37. A case in which a successful operation was performed in Christchurch Hospital in January, 1933, is therefore worth recording.

Clinical History.

Mrs. W., aged sixty-three years, was admitted to hospital on December 29, 1932, complaining of abdominal pain of twelve hours' duration, of nausea and flatulence. The pain began at 11 a.m.; at first it was in the right loin, but later it became generalized to the whole abdomen. Pain was also present in the thoraco-lumbar region. The patient had never had a similar attack of pain, but she had frequently suffered from flatulence and distension after meals. The bowels tended to be constipated. They had moved on December 28, 1932, and a little on the day of the patient's admission to hospital. At the time of admission pain was present to the left of the umbilicus in the upper part of the abdomen and in the lower part of the abdomen.

On examination the patient was seen to be an elderly woman in obvious distress and with an anxious expression. Her temperature was 35·8° C. (96·4° F.), her pulse rate was 97, and her respirations numbered 20 in the minute. She was complaining of severe pain in the epigastrium and definite tenderness and rigidity were present in that area. The liver dulness was definitely diminished.

An enema was given with good result, and the patient passed flatus freely. A leucocyte count revealed 12,000 to 14,000 cells per cubic millimetre; 88% of the cells were polymorphonuclear.

In view of the onset and site of the pain, in conjunction with the result of the leucocyte count, the diminished liver dulness and rigidity in the upper part of the abdomen, a provisional diagnosis of perforated gastric or duodenal ulcer was made.

Operation was undertaken. The abdomen was opened by a right paramedian supraumbilical incision, the rectus muscle being dissected from its sheath and retracted outwards. There was no sign of free fluid, but the first structure presenting was the lesser curvature of the stomach. There was no sign of perforation, but in the lesser sac above the level of the stomach was a thin-walled swelling, larger than the closed fist and covered by a thin layer of gastro-hepatic omentum. It was possible to distinguish a longitudinal band through the thinned out omentum, showing the swelling to be large bowel. Next the caecum was looked for, but the right iliac fossa was empty, and on tracing the terminal part of the ileum it was found to lead upwards towards the foramen of Winslow and to disappear. It was then clear that the swelling consisted of a small portion of the terminal part of the ileum, the caecum, ascending colon and hepatic flexure. The foramen itself admitted two fingers easily and three with slight difficulty.

Next the gall-bladder was explored and found to be normal; the common duct was identified, together with the remaining structures, portal vein and hepatic artery, forming the margin of the foramen. These structures could be picked up in one mass, and deep to them was the herniated bowel leading to the lesser sac. There was no element of strangulation present, the colour of the bowel wall being quite normal. Traction was not successful in reducing the contents.

Next the thinned out segment of bowel was punctured with a fine trocar, gas only escaping, after which by invagination the whole of the contents were returned to the abdominal cavity without difficulty. It was then found that the caecum, ascending colon, hepatic flexure and transverse colon had a complete mesentery, and it was possible to deliver the whole on to the abdominal wall. The bowel was replaced in its normal position, the caecum being returned to the right iliac fossa. The appendix was found completely retrocaecal, about 17·5 centimetres (seven inches) long and completely covered by peritoneum. It was removed by the usual purse-string technique. No attempt was made to reduce the size of the foramen, and the abdomen was closed in layers without drainage.

The patient, except for a transient distension which was relieved by an aperient, made an uninterrupted recovery, and left hospital on the eighteenth day.

Review of the Literature.

Alexander Miles, in Choyce's "System of Surgery", briefly reviews the condition.[1] He emphasizes its rarity and its salient clinical features of epigastric pain and a swelling being present, dull on light but resonant on heavy percussion; he also notes that there are no symptoms present due to pressure on the portal vein, hepatic artery or common bile duct, which run in the margin of the foramen.

William B. Coley,[2] in a review of the literature, notes that the first case was found at autopsy and was reported by Blandin in 1823. Rokitansky reported a second in 1842, Treitz a third in 1857, Wilson Moir a fourth in 1867 and Majoli a fifth in 1884.

The subject has been exhaustively treated by Moynihan[3] in "Retroperitoneal Herniæ", 1906, and more recently by Jeanbrau and Riche. The two last-mentioned ascribe some violent muscular effort in the production of the hernia.

According to Moynihan, the causes may be set out as follows: (i) a common mesentery for the whole of the intestine; (ii) absence of secondary fusion of ascending colon to posterior abdominal wall; (iii) an abnormally large foramen; (iv) an abnormal length of mesentery, giving rise to undue mobility of the intestine.

Ullmann,[4] in a complete review of the cases, brings the number published at that time to thirty; of that number of patients only eight recovered. He traverses the cases in detail, and reviews the mechanism of production, age period, contents, symptoms, diagnosis and treatment.

In regard to symptoms, in nine out of twelve cases collected by Jeanbrau and Riche there was epigastric or peri-umbilical tumefaction. In thirteen cases epigastric pain and tumour, as described by Alexander Miles, were present.

Nat H. Copenhaver, in the "Collected Papers of Mayo Clinic",[5] reviews the literature, and finds 23 cases, with one case from the Mayo Clinic. In this case the terminal part of the ileum, the caecum and the ascending colon had herniated through the foramen of Winslow into the lesser sac and then out through the gastro-hepatic omentum into the general peritoneal cavity. He again calls attention to the difficulty of reduction owing to the structures in the margin of the foramen.

Rowlands and Turner[6] note the difficulty of diagnosis before a laparotomy has been performed, but cite Sherren's case as having been diagnosed before operation. They also emphasize the difficulties of reduction and of stretching of the foramen, and quote Treves's case in which reduction even at the necropsy was not possible until all the structures—bile duct, portal vein and hepatic artery—had been severed. They agree with Moynihan that the sac should be opened anteriorly and reduction of the bowel from within attempted.

In regard to treatment, writers emphasize reduction of the condition on its being found. Some advocate the enlargement of the foramen by various means. Moynihan is directly opposed to any such procedure, holding that it is quite impossible in the living subject; and he advocates opening the lesser sac through the gastro-hepatic omentum and opening the distended coil of bowel, after which gentle traction is sufficient to reduce the hernia.

It is not considered necessary to attempt to reduce the size of the foramen, as recurrence is unlikely to take place here.

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(1) Alexander Miles: Choyce's "System of Surgery", 1925, Volume ii, page 488.

(2) William B. Coley: "Keen's Surgery", 1910, Volume iv, page 103.

(3) Berkeley Moynihan: "Retroperitoneal Herniæ", 1906.

(4) F. Ullmann: "Hernia Through the Foramen of Winslow", *Surgery, Gynecology and Obstetrics*, Volume xxxviii, 1924, page 225.

(5) N. H. Copenhaver: "Intraabdominal Hernia", "Collected Papers of the Mayo Clinic", Volume xv, 1923, page 322; *Archives of Surgery*, Volume vii, 1923, page 332.

(6) Rowlands and Turner: "Operations of Surgery", 1927, Volume ii, page 345.

NEUROFIBROMATOSIS ASSOCIATED WITH PROPTOSIS AND DEFECT OF THE ORBITAL WALL.

By A. Eisdell Moore,
Auckland.

Several cases of proptosis associated with neurofibromatosis have been recorded, and Le Wald in 1933 drew attention to a defect in the bony orbital wall that might occur in these cases. The occurrence of two such cases coming under the care of one surgeon in the course of a few months warrants publication.

Figure I.

Case I.

The first case was that of a child, aged seven weeks, referred on April 3, 1934, on account of vomiting and loss of weight. The child had shown a proptosis at birth, and had been under the care of Mr. Fairclough, who had enucleated the left eye for congenital glaucoma. A diagnosis of pyloric stenosis was established, and Rammstedt's operation was performed under gas and oxygen anæsthesia.

At the time of operation the following notes were made regarding the patient's face: "The left eye is absent, having been enucleated for congenital glaucoma. The left side of the face shows some slight paresis. There is an indefinite nodular swelling in the soft tissues of the left cheek running roughly in the line of Stenson's duct, but there is no swelling of the parotid."

The child made a good recovery from the operation, and steadily put on weight.

Soon after the operation the child was shown at a meeting of the Auckland Clinical Society, and the consensus of opinion was that the swelling in the cheek was in the nature of a cystic hygroma or, at any rate, of lymphatic origin. Dr. Coverdale, however, said that there was an established association between congenital glaucoma and neurofibromatosis, and suggested that the subcutaneous swelling might be due to such a cause.

The child was not seen again till November, 1934, when it was sent for because of the patient described in Case II having come under observation. The change was amazing. The whole of the left side of the head was greatly enlarged, and there was a soft swelling occupying the temporal region with nodular infiltration of the upper lid and the left side of the forehead. The face drooped on the affected side, but there was no actual paralysis.

FIGURE II.

The radiologist's report was as follows: "There is absence of bone detail of the outer wall and part of the roof of the left orbit. No orbital fissure or optic foramen can be demonstrated. The left orbit is enlarged. The findings are those of congenital bone absence." (See Figure I.)

On December 19, 1934, the nodular subcutaneous swelling over the forehead was removed and found to be a neurofibromatosis of the supraorbital nerve. The greatly thickened stem of the mass in the cheek was also removed, although it was impossible to dissect free all ramifications. This stem was undoubtedly the subcutaneous lump noticed at the performance of the Rammstedt's operation.

Both specimens were reported by the pathologist to be typical neurofibromata. Figure II shows the child's appearance at this date.

On examination on January 22, 1935, the deformity was apparently slowly increasing. The head of the child, practically a year old, had a circumference of 49·3 centimetres (nineteen and three-quarter inches), the normal being 45 centimetres (eighteen inches) at this age, and this increase was entirely on the left side.

Case II.

The second patient was referred to me by Dr. Coverdale on October 15, 1934. This was a child, aged four years, with the history that the left eye had been cloudy at birth and distinctly larger than the right. Dr. Wales, who saw the child at that time, has reported to me that he made a diagnosis of infantile

FIGURE III.

glaucoma, and although trephining was considered, it was thought best to treat the condition with eserine and bandaging. After treatment for three years the eye was still growing and was operated upon in Christchurch in 1933. Enlargement and asymmetry of the head had been noted within one month of birth.

On examination there was a small sclerosed blind eye. The left upper lid was full and pendulous, and there was an extensive subcutaneous tumour

of the left side of the forehead. As this lump was increasing rapidly in size, operative removal was recommended.

X ray examination at this stage showed: (i) Relative enlargement of the left orbit. The transverse diameter was 4·2 centimetres compared with 3·3 centimetres on the right side. The height was 5·0 centimetres as against 4·3 centimetres on the right side. (ii) Upward displacement of the lesser wing of the sphenoid. (iii) Enlargement of the pituitary fossa. (iv) Absence of the left anterior clinoid process. (v) A relatively small left maxillary antrum. (vi) A possible absence of part of the inner wall and roof of the left orbit. (vii) Irregular absorption of the zygomatic bone. (viii) Bulging of the left parieto-temporal region. The width of the sagittal plane was 10·5 centimetres on the left side as against 8·0 centimetres on the right side. (See Figure III.)

The tumour removed at operation was a plexiform neurofibroma, arising from the supra-orbital nerve.

Figure IV shows the child's general appearance with asymmetry of the head.

FIGURE IV.

Discussion.

These two cases both show three salient points: (i) proptosis, (ii) neurofibromatosis, (iii) congenital absence of the orbital wall.

The first two of these symptoms have been associated for many years. In the article on neurofibromatosis in Choyce's "System of Surgery",[1] it is stated that "plexiform neurofibroma may occur with some degree of proptosis and is usually associated with characteristic swelling of surrounding parts".

R. Foster Moore,[2] writing on neurofibromatosis with proptosis, describes such a case in a man of thirty-three years whose eye was normal at birth, but proptosis then began and was associated with pulsation, for which the carotid had been tied without benefit. The skiagram in this case showed an abnormally large orbit and a deficiency in the posterior orbital wall providing a free communication between orbit and cranial cavity, and Moore considered the pulsation to be a direct transmission from brain to orbital tissues.

Rockliffe and Parsons[3] published the case of a child of two years who died after orbital clearance and carotid ligation. The whole bone of the orbit except the floor and outer wall had disappeared or had never developed.

Leon Le Wald[4] recently described four cases of congenital absence of the superior orbital wall associated with pulsating exophthalmos, and in at least two of these cases there was evidence of neurofibromatosis. One of these patients was successfully operated upon by reforming the wall of the orbit by a bone transplant from the skull. Le Wald lays stress on the necessity of distinguishing these cases from sarcoma of the orbit, and points out that the X ray appearances are sufficiently characteristic to make the diagnosis clear.

Dr. Bruce Mackenzie's report on the X ray appearances in Case II brings out all the important diagnostic points.

In the two cases described in this article the eye had had to be operated upon at an early date and pulsation had not been recorded.

References.

(1) Choyce: "System of Surgery", Volume i, page 46.

(2) R. Foster Moore: "Diffuse Neurofibromatosis with Proptosis", *British Journal of Ophthalmology*, Volume xv, May, 1931, page 272.

(3) Rockliffe and Parsons: *Transactions of the Pathological Society of London*, 1903.

(4) Leon Le Wald: "Congenital Absence of the Superior Orbital Wall Associated with Pulsating Exophthalmos: Report of Four Cases", *American Journal of Roentgenology*, December, 1933, page 756.

The Australian and New Zealand Journal of Surgery.

All articles submitted for publication in this journal must be typewritten and double or treble spacing should be used. Each article should conclude with a brief summary and statement of conclusions. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without any abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal together with that of the journal in which the abstract has appeared, should be given, with full date in each instance.

When illustrations are required, good photographic prints on glossy gaslight paper should be subm'tted. Line drawings, charts, graphs and so forth should be drawn on thick white paper in India ink. Authors who are not accustomed to prepare drawings of this kind, are invited to seek the advice of the Editor if they are in any doubt as to the correct procedure. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available.

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HYDATID DISEASE AND HOSPITAL RECORDS.

THE annual meeting of 1935, though chiefly memorable for the brilliant functions attending the opening of the College home in Melbourne, was also of exceptional interest from the educational and scientific points of view. Amongst the various subjects dealt with, hydatid disease rightly took a prominent place.

It is the desire of the Council to foster the study of this malady with a view to the acquirement and publication of much needed information concerning its pathology and treatment, and also to combat what may possibly become a serious menace to the bodily and industrial welfare of our people.

Australia and New Zealand are the only English speaking countries in which hydatid disease is at all prevalent, and ought to be regarded as centres of authority and as guides, at least for the British Empire, in the subject of hydatid disease.

Australasian surgeons have contributed freely on the clinical side to the world's hydatid literature, and some valuable laboratory work has been done, particularly at the Walter and Eliza Hall Institute of Medical Research in Melbourne by Dew, Kellaway, the Fairleys and

others; and recently the nucleus of a department for hydatid disease research and prevention has been inaugurated at the Medical School in Dunedin.

Argentine and Uruguay are blessed or cursed, according to how one looks at the matter, with a greater prevalence of hydatid disease than we have, and our colleagues there are making worthy efforts to gain and distribute information that will be useful in its prevention and better treatment. The first number of a magazine devoted entirely to hydatid disease has just appeared under the editorship of Dr. Fontana, of Montevideo.

A notable move made by the Royal Australasian College of Surgeons is the establishment of a Hydatid Registry of cases occurring in Australia and New Zealand. It is expected that within a year or two records of at least 1,000 such cases will be available for reference at the College Library. The usefulness of these records will of course be greatly enhanced if they are taken out in conformity with the case-taking forms adopted by the College. All hospitals on request can be supplied with these forms; and it is hoped that all surgeons will see to it that their hydatid case records contain as far as practicable the details asked for in these forms, and that a copy of these records is sent for filing in the College Registry.

Case records should be complete as well as concise. Some house surgeons make their case notes conscientiously and well, but some do not, especially those whose notes are not read by their chiefs or by anyone else who matters. In most of our large hospitals the filing of case records is done well, but the quality of the records themselves leaves much to be desired. The defects, which are chiefly those of omission, can be remedied by routine supervision.

Sir Holburt Waring, in speaking on the subject of surgical education at our recent annual meeting, said that the English College, of which he had the honour of being President, would not recognize a hospital as worthy of approval unless the case recording system was thoroughly efficient.

Cancer records, and in some hospitals goitre records, have been greatly improved of late years because special interest is taken in their study, and similar improvement is now hoped for in hydatid records.

Fellows of the College will render a useful service to surgical progress if they exert their powerful influence in the direction of securing better quality in case records.

L. E. BARNETT.

Surgery in Other Countries.

[In this column will be published short résumés of articles likely to be of practical value from Journals published in other countries and not readily accessible to surgeons in Australia and New Zealand.]

An Apparatus for Use with an Artificial Anus.

Kirschner (Heidelberg): *"Neuer Verschlusapparat für den kunstlichen Bauchafter"*, *Zentralblatt für Chirurgie*, 1935, Number 10, page 580.

Kirschner discusses, from the point of view of cleanliness, the disadvantages of the average apparatus designed for a patient with an artificial anus. For his patients Kirschner uses a belt constructed in the following way:

A broad belt of thick rubber, obtained from an old motor tube, is fitted to the front of the abdominal wall. The belt is held firmly over the artificial anus by straps which go round the upper part of the thigh. In the belt is an opening over the artificial anus, which is covered by a hinged rubber flap, the flap being fastened by a press stud or "zip". Under the flap the opening in the belt is padded with cellular material.

On feeling pressure from the fæces, the patient leans forward, opens the flap and, without soiling himself, empties his bowel into an ordinary sanitary arrangement. The patient then inserts fresh cellular material, replaces and fastens the flap. By using this belt, thick fæces can be retained in the bowel for a considerable time.

H. B. Devine.

The Treatment of Cardiospasm.

Professor H. Starck (Karlsruhe): *"Die Behandlung des sogenannten Kardiospasmus (Kardiotonische Speiseröhrenerweiterung)"*, *Der Chirurg*, October 15, 1934, page 697.

Professor Starck outlines his method of treatment of what he, in agreement with Hurst, regards as an achalasia of the cardiac sphincter. He has devised a special dilator for the purpose of stretching the cardiac ring. Its shaft is narrow and elastic, and has at the distal end a dilating mechanism consisting of four bars, six centimetres long and capable of dilating to a circumference of twelve centimetres. Distal to the dilator is a detachable director. This consists of a rubber tube twenty-five centimetres long and filled either with mercury or shot. The instrument, when passed into the œsophagus, seldom fails to find its way through the cardiac orifice. In difficult cases the use of different standing and lying positions will induce the director to pass. The dilator is smeared with paraffin and the distance from teeth to diaphragm is marked on it. It is passed with the patient sitting with the head back, and when it is in position the handles are compressed suddenly two or three times with full force, and then withdrawn. A sensation of tearing should be experienced, and unless this is appreciated the procedure should be repeated. The tissue torn is the ring musculature only, and the mucosa escapes injury.

Patients are instructed to take full meals the day after treatment, and on succeeding days the stomach tube is passed to ascertain that the stomach is empty. If it is not empty or if there is still difficulty in swallowing, the dilator should be used again. Success is recognized when there is no longer any feeling of resistance to the dilator action.

The follow up of a large series of cases appears to show that this is a very satisfactory and enduring procedure. In preliminary investigation cases in which there is no anatomical obstruction should be eliminated, as they do not give good results.

ARTHUR E. BROWN.

A NEW SUTURE MATERIAL.

Richard Bertlesmann: "*'Carnofil': Ein brauchbares neues chirurgisches Nahtmaterial*", *Zentralblatt für Chirurgie*, 1935, Number 10.

HERR PAUL BOST has produced a new absorbent suture material, called "Carnofil", to be used instead of catgut. It is produced from the fibre of horse muscle, which, unlike the intestine of the sheep, is clean material in its primary state. During the past year it has been carefully tested by the author in Red Cross work in Kassel.

Bertlesmann's observations are as follows: (i) When properly treated, the "Carnofil" thread is completely sterile. (ii) Its tensile strength is equal to that of catgut. (iii) In the body it is absorbed in the time period designed by its preparation. (iv) It is rougher than catgut, especially in the thicker numbers.

The manufacturers, however, are now making the finer numbers of "Carnofil" smoother, and they have been found quite satisfactory for intestinal sutures in gastric resections. Bertlesmann made many thorough bacteriological examinations of the new material, and the results, compared with those of "ready for use" catgut, were satisfactory. In conclusion, the author considers that "Carnofil" has the following two advantages: (i) The original material from which it is made is clean. (ii) It is plentiful in Germany and thus renders the country independent of outside suppliers. At present, Germany does not permit the export of "Carnofil".

H. B. DEVINE.

HYDATID CYSTS RUPTURED INTO THE PERITONEAL CAVITY AND THE RESULTING MEMBRANES OF ENCYSTMENT.

Velarde Perez Fontana (Professor of Surgical Pathology, University of Montevideo): "*Quistes hidatidicos rotos en el peritoneo y las membranas consecutivas de enquistamiento*", Montevideo, 1932.

IN this comprehensive and well documented monograph, Fontana discusses the milestones of our knowledge regarding intraabdominal rupture of abdominal cysts, and he criticizes adversely in some minor points the views advanced by Dévé, Dew and others. He supports Dévé, Fairley and Wright, and Lagos Garcia in attaching prime importance to the scolex in the formation of secondary cysts of all kinds, whether inside or outside the parent cyst. He considers that rupture occurs more commonly in the case of univesicular than in multivesicular cysts.

He describes the possible results of rupture that may occur under varying circumstances:

1. Escape of hydatid fluid with toxic and sometimes anaphylactic manifestations.
2. Hæmorrhage from the vessels in the lacerated pericyst.
3. Heterotopic cyst formation from the escape into the peritoneal cavity of the mother cyst or of its daughters.
4. Escape of scolices with result of multiple secondary cyst formation.
5. Pseudotubercular nodules, the result of abortion and involution of some of these cysts.
6. Escape of bile more or less continuously from the lacerated liver cyst. If it is small in amount, this bile may be completely absorbed. If the amount is large it leads to the condition known as choleperitoneum. The bile does not interfere with the development of daughter cysts, but it irritates the peritoneum and leads to the formation of a false membrane that glues the viscera together.

Lipoid degeneration may occur simulating chylous ascites. A point in differential diagnosis here that is worth remembering is the usual presence of lumbar resonance on percussion in the hydatid form of effusion. Pyogenic and, in rare cases, pyopneumogenic infection may complicate the clinical picture.

7. The false membrane of encystment and the changes that it may undergo are fully described. It is a fibrino-cellular layer showing fibrosis in its deep layers and the special features of minute deposits of bile pigment and rudimentary or aborted hydatid elements and giant cells. Apart from the bile staining, Fontana holds that a very similar false membrane may result from the intraperitoneal rupture of a clean cyst of liver or even of a cyst of the spleen or omentum. A large series of photomicrographs illustrates the description of the false membrane.

L. E. BARNETT.

WHITE MICE AND EXPERIMENTAL ECHINOCOCCOSIS.

F. Dévé: "*L'echinococcose expérimentale de la souris blanche*", *Archivos Internacionales de la Hidatidosis*, December, 1934, pages 85-94.

DÉVÉ is continuing hjs investigations on the efficiency of various alleged remedies for hydatid cysts (serums, anatoxins, drugs *et cetera*), and is now using for this purpose white mice. He finds that these animals have many advantages over the commonly used rabbit. In particular, the intraperitoneal injection of hydatid sand is almost certain to be followed by the desired cyst formations, whereas a like result is very chancy in the rabbit, and takes a much longer time. Moreover, mice are cheap and easy to feed and house.

Dévé in this short but very instructive paper makes a valuable contribution to the quest for a systemic or general cure of hydatid cysts.

L. E. BARNETT.

THE SURGICAL TREATMENT OF CHRONIC AND EXTENSIVE DUODENAL ULCER.

Professor H. Finsterer: "*Resektion zur Ausschaltung oder Gastro-Enterostomie beim nicht resezierbaren Ulcus Duodeni*", *Zentralblatt für Chirurgie*, 1934, Number 28, page 1634.

IN this paper, Finsterer discusses the surgical treatment of chronic duodenal ulcer, and particularly that type which is so chronic and widespread that it cannot be resected. He also discusses the advantages and disadvantages of the operations used for this condition, namely, gastro-enterostomy, the operation in which a duodenal ulcer with two-thirds of the stomach is removed (radical operation), and the operation of exclusion with resection. On the Continent, gastro-enterostomy as a surgical treatment of chronic duodenal ulcers has few adherents. It is, as a rule, employed in the treatment of old people, where the general condition of the patient is too bad to allow the carrying out of the radical operation of exclusion with resection. In most of the Continental clinics the radical operation has come to be the one preferred; but, as Finsterer points out, it carries with it dangers for the patient; it is true, however, that the permanent results are good—as high as 95%.

Resection of a chronic duodenal ulcer which has perforated the pancreas, is technically difficult. The mortality from this operation will depend upon the skill and experience of the surgeon, and is high even in the best circumstances. There are a number of such chronic duodenal ulcers which cannot be resected. Statistics taken from the various German clinics show that the number of these unresectable ulcers varies from 6% (von Haberer) to 50% in country hospitals. In Finsterer's own clinic, the unresectable chronic duodenal ulcers amounted to 16%, but in the last five years they were only 7%. The great danger of the radical operation lay in the dissection of the ulcer from the vital structures in its neighbourhood, and in the difficulty of properly suturing the duodenal stump, in which, under such conditions, suture insufficiency must frequently occur.

Sixteen years ago Finsterer suggested an operation in which he advocated the partial resection of the stomach, combined with the exclusion of the duodenal ulcer, by closure either distal or proximal to the ulcer. This operation he called the exclusion with resection operation (*Resektion zur Ausschaltung*). Its effect was twofold: first, the chronic ulcer was removed from the action of the acid gastric juice, and from the irritating action of the food, and, therefore, it was thought that it should heal very quickly; secondly, the occurrence of jejunal ulcer was minimized by an abundant resection of the acid-forming part of the stomach.

A retrograde filling of the duodenum sometimes interfered with the healing of the excluded duodenal ulcer. In more recent years, Finsterer avoided this complication by using Hofmeister's modification of the Reichel-Pólya gastro-intestinal method of anastomosis. Figure I illustrates this Hofmeister-Finsterer type of anastomosis.

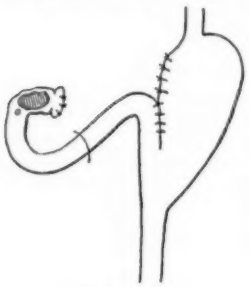

FIGURE I.

The exclusion with resection operation, Finsterer thought, avoided the difficulties and dangers of the "radical" operation, because the ulcer was not disturbed.

The early operations of exclusion with resection were not quite satisfactory, owing to the fact that in the earlier years Finsterer was inclined to remove only about half of the stomach. He soon found that, in order to obtain healing of the duodenal ulcer and also to avoid the occurrence of jejunal ulcer, it was necessary to remove two-thirds of the stomach; that is, as much of the acid-bearing mucous membrane must be removed as in the case of a radical resection.

This operation of exclusion with resection did not become popular with surgeons, for the reason that, as so many jejunal ulcers recurred in the operation of pyloric exclusion (von Haberer, 17%; Clairmont, 27%), it was thought that the pylorus and the antrum (*Zurucklassen des Pylorus*) were big factors in causing hyperacidity. Its unpopularity was also due to the fact that surgeons did not remove enough of the stomach to reduce the gastric acidity, and consequently, in a high percentage of cases, their patients developed jejunal ulcer.

In some cases it was found that the occurrence of jejunal ulcer was due to the fact that in the case of a very dilated stomach, two-thirds of the stomach had not been removed, but only what appeared to be two-thirds. It is not a question of how much is taken away, but of how much is left; that is, only a third of the stomach should be left.

The accompanying table shows the result of the exclusion with resection operation in regard to permanent cure and to the occurrence of jejunal ulcer.

Operation.	Surgeon.	Result.	Number of Cases.	Jejunal Ulcer.
Exclusion with resection without removal of pylorus.	Finsterer.	Of those patients operated on for over three years, 90·3% were trouble-free.	81	6·1%; three cases were in 1919, when inadequate amount of stomach was removed; 3% of jejunal ulcer in operation, as he now performs it.
Exclusion with resection without removal of pylorus.	Finsterer.		Last ten years, 33 cases. (Modern exclusion with resection operation in which an adequate amount of stomach had been removed.)	One case = 3%.

In regard to the operation of exclusion with resection, there has been much controversy concerning the level at which the exclusion should be made. If it was made proximal to the pylorus, the closure was easy and suture insufficiency never occurred. The objection to this procedure was the retention of part of the antrum and the pylorus. It was thought that this retention caused hyperacidity, and therefore was a cause of jejunal ulcer. The closure of the divided duodenum, with the chronically inflamed duodenal ulcer in the neighbourhood, was difficult, and suture insufficiency, with its result—peritonitis—was likely to occur. The mortality of the operation, when the exclusion was made at this level, was therefore much greater. In order to avoid the effect of the mucous membrane of the retained antrum, Wilmanns suggested that it should be excised before closure of the aboral gastric stump ("mucoclasis").

Judging from his own experience over the last sixteen years, and from the statistics of many large German clinics, Finsterer is of the opinion that the same permanent curative results of the operation of exclusion with resection are obtained whether the pylorus is removed or not. He thinks that exclusion with resection, with the exclusion at a pre-pyloric level, is an operation with only 4% of mortality, and one, therefore, which he can recommend to the average surgeon as giving a permanent cure in 94% of cases of chronic unresectible duodenal ulcers.

Finsterer then discusses the advantages of the exclusion with resection operation over gastro-enterostomy. He points out that gastro-enterostomy is an operation which, on the Continent, is only done in special circumstances—where the patients are old, where the general condition of the patient is too bad to permit any other operation, in cases in which the perforation is associated with stenosis, or sometimes where there is a bad bleeding. As a consequence of this, the immediate and permanent results of gastro-enterostomy are not as good as those of exclusion with resection. The conclusion is not quite justified, for gastro-enterostomy is an operation which, on the Continent, is only used as an emergency operation in very debilitated cases. The principle of its application is therefore entirely different from that employed by the English and the American surgeon.

H. B. DEVINE.

HYDATID CYSTS OF THE SPLEEN.

L. Sabadin (Algiers): *"Les kystes hydatiques de la rate"*, *Journal de Chirurgie*, Volume xlv, April, 1935, page 534.

THIS careful study of hydatid cysts of the spleen by Sabadin comes from the surgical clinic of Professor Costantini of Algiers, and is based on two cases personally observed by the author, ten cases previously unpublished (communicated by his colleagues), and two hundred that he has garnered from the literature.

The historical items, comparative statistics and the pathological and clinical details are definitely valuable for reference purposes, and here and there add something to our practical knowledge of splenic invasion, although, as is well known, Dew's work covers this subject fairly thoroughly.

Only 2% or 3% of the cysts occurring in man are located in the spleen, and some of these are secondary, owing their origin to a leaking or ruptured liver cyst.

The great majority of splenic cysts grow downwards towards the pelvis and remain latent for many years; a few grow upwards into the thorax and produce symptoms much earlier.

The invaded spleen always becomes adherent to adjoining structures, sometimes very adherent. Splenectomy therefore as a method of treatment is rendered difficult and dangerous. The author strongly advises against splenectomy as a routine procedure, and recommends marsupialization wherever possible. Splenectomy has a mortality of 20·5%, marsupialization 9·4%. Evolution is slowly progressive, and the cyst may reach an enormous size. There are rare instances of spontaneous cure from obsolescence and calcification, or from evacuation into

the neighbouring colon or stomach. Bronchial vomica also occurs, but never with cure resulting. Rupture into the peritoneal cavity is not uncommon and may lead to secondary dissemination. Ruptures into the renal pelvis or through the skin are surgical curiosities. Suppuration, usually of low grade, occurs in about 50% of cases.

The section dealing with diagnosis is well handled, and opens with the impressive dictum: "Always regard swellings in the left hypochondrium with a hydatid suspicion."

Cases from Mustapha Hospital are as follows: (1) A man, aged forty-two years, had been treated medically for years. In July, 1933, he had a sudden attack of violent pain. Rupture of a cyst was suspected. Operation was performed next day. A huge intact cyst weighing 4·8 kilograms was found extruded through a rent in the enlarged spleen, and within the organ was another cyst the size of an orange; no daughter cysts were found. Marsupialization was performed with drainage; a smooth recovery resulted. (2) A woman, aged fifty-two years, had an operation in July, 1933, for a splenic cyst *plus* a recurrence in a scar following operation for a liver cyst three years previously. Two large cysts at the lower part of the spleen were evacuated and drained separately; the contents were purulent fluid and daughter cysts. The recurrent grafted cyst was intramuscular and easily excised; good recovery followed.

Previously unpublished cases communicated by Dr. Oulié of Constantine are as follows: (1) A woman, aged twenty-three years, had an operation in July, 1933. Three large cysts were noted in the spleen. Splenectomy was performed; much difficulty was experienced from hæmorrhage and firm adhesions. Good recovery followed. (2) A man, aged twenty-five years, was operated on in November, 1933. A deep seated cyst in the spleen was evacuated (three centimetres); two litres of clear fluid and mother membranes were removed. No daughter cysts were found. Free bleeding occurred. Marsupialization and drainage were carried out. A stormy convalescence followed and a chronic sinus persisted.

The following cases were communicated by Dr. Guedj: (1) A woman, aged thirty-seven years, had an operation in January, 1933, for a large univesicular cyst (three litres). Marsupialization and drainage were carried out; slow but good recovery followed. (2) A woman, aged thirty years, had an operation in August, 1933. A huge cyst containing 15 litres of fluid, and three daughter cysts were evacuated from the upper pole of the spleen. Very extensive adhesions were present, but nevertheless splenectomy was carried out; suppuration and death followed. (3) A woman, aged twenty-five years, had an operation in April, 1934. A large simple cyst was evacuated, and the wound was closed without drainage. One subsequent tapping was necessary; good recovery followed.

The following cases were communicated by Dr. Revel: (1) A woman, aged twenty-seven years, had an operation in October, 1931. A large simple cyst was evacuated and closed without drainage. Suppuration occurred and drainage was established. Chronic sepsis followed, and death occurred ten months after the first operation. (2) A woman, aged twenty-five years, had an operation in May, 1932. A large cyst with daughter cysts was marsupialized and drained; good recovery followed.

The following cases were communicated by Dr. Engel: (1) A man, aged thirty-eight years, had an operation in September, 1932, for a simple cyst the size of a child's head. Marsupialization was performed and good recovery followed. (2) A woman, aged forty-five years, had an operation in October, 1932. A simple cyst the size of a fœtal head was evacuated and closed without drainage; a good result followed.

L. E. BARNETT.

Reviews.

HYDATID DISEASE OF BONE.

Hidatidosis Osea. By Oscar Ivanissevich; 1934. Buenos Aires. Imprenta amorrortu; pp. 246.

This book of 246 pages in large quarto size, handsomely printed, generously illustrated regardless of expense, and profusely documented with clinical, pathological, radiological and bibliographical references, represents a vast amount of painstaking effort. The author is certainly to be congratulated on a fine piece of work that summarizes and brings up to date the world's knowledge of hydatid disease as it affects bone. Not that this knowledge is by any means perfect yet: we are, for example, still in doubt as to whether, in exceptional cases, hydatid cysts originating in the soft parts can invade and infiltrate bone, and we still lack an explanation for the occasional spread from one bone to an adjoining bone without apparent involvement of the joint between them. The author, in discussing these and other problems, assumes the presence and action of scolices, but, according to Dévé, scolices in bone cysts are, as a rule, absent.

In the Argentine Republic the author has been able to collect records of 75 cases—27 of them not previously published and 10 of them coming under his own personal observation in Professor Arce's clinic during the last fifteen years. The estimated incidence of bone invasion is 2% of all hydatid cases, and the locations noted are: vertebræ, 18 cases; pelvic bones, including sacrum, 18; humerus, 12; tibia, 10; femur, 9; ribs, 2; scapula, 1; radius, 1; sternum, 1; cranium, 1; bones of face, 2. The localization of hydatid cysts is dependent to a considerable extent on biological peculiarities and affinities, many of them very imperfectly understood at present. The author stresses the point, for instance, that bone cysts are rarely, if ever, seen in animals.

Attention is called to the slowness, the gentleness and the latency of bone invasion, so that it may be many years before any clinical signs become manifest, and only when some complication, such as pathological fracture or infection, takes place is the patient brought under medical observation. The quiet insidious process of bone infiltration is not accompanied by any obvious inflammatory reaction, and within the bone there is no formation of an adventitia. The cortex of the bone is not thickened, nor is it distended by the contained cysts, although they may be in great quantity and in long bones reach from one end to the other. Pathological fracture is not uncommon, and, according to the author, bony reunion may occur in spite of the continued presence of cysts. The true alveolar or Bavaro-Tyrolean type of hydatid disease has scarcely ever been seen in bone—only three or four cases are on record. Once the cysts have spread beyond the confines of the bony cortex, growth becomes more rapid and assumes the ordinary type of exogenous development. What Dévé has named the chronic ossifluent hydatid abscess or pseudo-abscess, results from an agglomeration of extraosseous cysts that have undergone degeneration, maceration and partial liquefaction. It is seen most commonly in connexion with hydatid disease of vertebræ, and may be mistaken for a chronic tuberculous abscess associated with Pott's disease of the spine.

In diagnosis the author has found laboratory aid rather disappointing. In 31 cases of proved bone infection the complement fixation test gave a positive reaction in only 60%, and the Casoni skin reaction was obtained in 59%. Eosinophilia to a greater degree than 5% was present in only 36% of these cases. X ray examination is, of course, helpful, but only in the later stages of bone

invasion or in extraosseous development. In vertebral cases when intrathecal extension has taken place, lipiodol injection may give some localizing information. Differential diagnosis has to be made from other bone affections due to tuberculosis, syphilis, osteomyelitis, angioma, chondroma, sarcoma, von Recklinghausen's disease and *osteosis fibrogeodica*. The author suggests that radiological research should be undertaken with a view to discovering some dye that will enable hydatid membrane to be easily demonstrated in an X ray film.

Treatment is briefly described under the headings of: (i) Curettage, which must be meticulous and may have to be repeated. (ii) Excision of the affected bone or part of it; bone grafting may be usefully employed after this procedure. (iii) Amputation, especially when joint invasion has taken place.

Ivanissevich's book is a valuable addition to hydatid literature, and usefully supplements the excellent account of bone cysts that is given in Professor Harold Dew's well-known book, "Hydatid Disease", published in Sydney in 1928.

A NEW JOURNAL ON HYDATID DISEASE.

Archivos Internacionales de la Hidatidosis. Departmento Cientifico de Salud Publica: Montevideo. Volume i, Fasciculus 1, December, 1934. Pp. 135, with 12 illustrations.

IN the Republic of Uruguay there has been established a centre for the study and prevention of hydatid disease, and the first number of a journal devoted solely to these purposes has just been issued under the editorship of Dr. Velarde Perez Fontana, Professor of Surgical Pathology at the University of Montevideo.

Dr. Fontana, in his editorial article, tells us that the spread of the disease in Argentina, Uruguay and Brazil has occasioned much alarm, but that this has not yet been followed up by effective action on the part of the sanitary authorities. Duly appointed medical commissions have certainly drawn up reports and recommended preventive measures, but to a large extent these recommendations have been pigeon-holed.

The visit of Professor Dévé to South America in 1932, the comprehensive course of lectures that he then gave, and the hydatid exhibition that was organized at this time, focused public attention on the gravity of this national malady, and, as a result, a centre for the study and prevention of hydatid disease has been established at Montevideo by the Minister of Public Health. This centre is to work in conjunction with a similar centre at Buenos Aires, under Arce, Cranwell and Vegas, and willing support from other countries has been indicated by Dévé (France), Putzu and Pinelli (Italy), Lozano (Spain), Einarsson (Iceland), Dew (Australia), and Acland and Barnett (New Zealand).

Compulsory notification of hydatid disease has been decided upon in Uruguay, and New Zealand is held up as an example of the usefulness of legislation in this direction. This is unfortunate, for, although it is true that hydatid disease was made notifiable in New Zealand many years ago, the regulation has become practically a dead letter, owing to the negligence or indifference of medical practitioners and the health authorities. It is to be hoped that the Republic of Uruguay will have more satisfactory results from this so-called compulsory notification.

The statistics that have been collected in New Zealand for the last forty-three years are based on the public hospital records and the mortality tables as issued by the Government Department of Statistics.

The new journal contains a wealth of interesting matter including: (i) An account of the important contributions to hydatidology of Dévé, Posadas, Cranwell, Vegas, Lozano, Dew, Escudero, Castex, with short biographies and portraits. Others will be published in later editions. (ii) Comprehensive statistical articles dealing with incidence and mortality by Lomba and Pazos. (iii) Schemes and schedules for research and for a campaign of prevention. (iv) Special articles by noted

authorities, including one on the usefulness of white mice in hydatid experimentation by Dévé, and one on a fatal case of anaphylaxis by Lozano.

This journal will undoubtedly prove a very valuable addition to hydatid literature.

NERVE SUTURE.

The Conduct and Fate of the Peripheral Segment of a Divided Nerve in the Cervical Region when United by Suture to the Central Segment of Another Divided Nerve. By SIR CHARLES BALLANCE (from the Research Laboratories of the Royal College of Surgeons); 1934. London: Macmillan & Co., Ltd. Demy 4to, pp. 45, with 59 illustrations.

SIR CHARLES BALLANCE describes briefly in this publication the changes which take place in the neurilemma, myelin sheaths and axones of a peripheral nerve trunk when it is subjected to complete transverse section. From his studies he emphasizes the fact that following anastomosis by suture the histological characters of the regenerating fibres of a peripheral nerve are identical with those of the central segment from which their growth is proceeding. An examination of cell groups in the *medulla oblongata* of a cat in which the right recurrent laryngeal nerve had been sectioned, has suggested to the author that there are free neural connexions between the lower motor centres of the hind brain. The article is profusely illustrated.

WORK AT THE MAYO CLINIC.

Collected Papers of the Mayo Clinic and the Mayo Foundation. Edited by RICHARD M. HEWITT, M.A., M.D., and LLOYD G. POTTER; Volume xvi; 1935. Philadelphia: W. B. Saunders Company. Royal 8vo, pp. 1192, with illustrations.

EACH year the amount of work done in the Mayo Clinic increases, and it has become necessary to limit the "Collected Papers" by giving the title only of the vast majority. Of the 641 articles taken into consideration, 86 appear in full, 31 by abridgement and 459 by title.

Among the more important papers is one by Waltman Walters on the treatment of extensive malignant lesions of the stomach. He advocates exploration even if X ray examination reveals an apparently irremovable tumour; he has found that 15% of these can be removed.

Starr Judd, for duodenal ulcer, advises partial resection of the pylorus and ulcer, and a gastro-duodenostomy.

An interesting account of localized chronic ulcerative conditions of the small intestine, illustrating difficulties of diagnosis, is given by Brown and Bargen.

Charles Mayo writes on appendicitis and advises the Oschner treatment of peritonitis with various reservations. We note that he does not invaginate the appendix stump.

It is interesting to find that, even after the thorough investigation carried out at the Mayo Clinic, Buie finds that 71% of cases of high rectal pain are either undiagnosed or are labelled neurosis.

Balfour reports that of carcinomata of the alimentary tract which are subjected to operation, 50% are resectable. He recommends Devine's exclusion operation for inoperable carcinoma of the stomach.

Cabot contributes several articles on high acid treatment of urinary infections and believes it is of very definite value.

Ghormley and Henderson publish some interesting reviews of the end results of fracture therapy.

Adson summarizes his views of surgery of the sympathetic system.

A very large field is covered by the numerous other articles, and the high standard of work one expects from this clinic is well maintained.

CORRIGENDUM.

IN a review of "A Short Practice of Surgery", by Hamilton Bailey and R. J. McNeill Love, published in the tissue of October, 1935, at page 201, the price of the book was incorrectly stated. The price is thirty shillings net.

Notices.

SEVENTH AUSTRALIAN CANCER CONFERENCE.

THE Seventh Australian Cancer Conference, which is being convened by the Director-General of Health, Dr. J. H. L. Cumpston, C.M.G., will be held at the College, Spring Street, Melbourne, commencing on Monday, May 4, 1936, and concluding on Saturday, May 9, 1936. In due course, all members of the profession will receive a copy of the agenda for the conference, and the Council of the College hopes that all Fellows, who can conveniently do so, will attend.

NEW DEVELOPMENTS IN SURGICAL EQUIPMENT.

THE attention of Fellows is drawn to page xxvi among the advertisements, which illustrates some recent developments in surgical equipment. The Editorial Committee is responsible for the selection of the equipment illustrated thereon. In future, one or two pages of this nature will be included in each issue, and the publishers will be pleased, whenever possible, to supply the names and addresses of the manufacturers to anyone requiring such information.

THE BRITISH POST-GRADUATE MEDICAL SCHOOL.

THE College would like to draw the attention of readers of the journal to the announcement of the British Post-Graduate Medical School on page xxviii of the advertisements.

Editorial Notices.

EDITORIAL communications should be addressed to the Chairman of the Editorial Committee, 57 Collins Street, Melbourne, or to any member of the Editorial Committee. It is understood that original articles forwarded for publication are offered to THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY solely, unless the contrary be stated.

Reprints can be supplied at cost price; the minimum number is fifty copies. Orders for reprints must be given when the proof is returned.

Exchange journals should be addressed to the Honorary Librarian, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1, Victoria, Australia.

Business communications and remittances should be addressed to Butterworth and Co. (Aus.) Ltd., 8 O'Connell Street, Sydney.

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